

Date July 12, 1999

PATENT

Docket No. 16153-7775

CERTIFICATION UNDER 37 CFR 1.10

I hereby certify that this New Application Transmittal and the documents referred to as enclosed therein are being deposited with the United States Postal Service on this July 12, 1999 in an envelope as "Express Mail Post Office to Addressee" Mailing Label Number EL221795488US addressed to: Box Patent Application, Assistant Commissioner of Patents, Washington, D.C. 20231.

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NEW APPLICATION TRANSMITTAL

Transmitted herewith for filing is the patent application of

Inventor(s): William S. M. Wold, Karoly Toth, Konstantin Doronin and Ann E. Tollefson

For : Replication-Competent Anti-Cancer Vectors

Enclosed are:

1. Benefit of Prior U.S. Application (35 USC)

 The new application being transmitted claims the benefit of a and enclosed is added page for new application transmittal where benefit of a prior U.S. application claimed.

2. The Papers Required For Filing Under 37 CFR 1.53:

<u>40</u>	Pages of Specification
<u>1</u>	Pages of Abstract
<u>3</u>	Pages of Claims
<u>55</u>	Sheets of Drawing

 X formal informal

In addition to the above papers there is also attached:

<u>75</u>	Sheets of Sequence Listing
<u> X </u>	Disk Containing Sequence Listing
<u> X </u>	Statement of Content
<u> X </u>	Return Receipt Postcard
<u> </u>	Information Disclosure Statement with <u> </u> copies of references.

3. Declaration or oath

- ☐ Enclosed ☐ pages
- ☐ Newly executed (original or copy)
- ☐ Copy from a prior application (continuation/divisional with page 5 of 5 completed)
- ☐ Deletion of Inventor(s) (signed statement attached deleting inventor(s) of prior application)
- ☒ Not enclosed

4. Inventorship Statement

The inventorship for all the claims in this application are:

☒ the same

OR

☐ are not the same and an explanation, including the ownership of the various claims at the time the last claimed invention was made, is submitted.

5. Language

☒ English ☐ Non-English

A verified English translation of the

[check applicable item(s)]

☐ specification and claims

☐ declaration

is attached.

6. Assignment

☒ An assignment of the invention to Saint Louis University

☐ is filed under separate cover sheet

☐ was filed in the prior application

☒ will follow

7. Certified Copy

(Country)

(Application No.)

(Filed)

from which priority is claimed

☐ is attached

☐ will follow

8. Fee Calculation

CLAIMS AS FILED

	Number Filed	Provided with Basic Fee	Number Extra	Rate	Basic Fee \$760
Total Claims	27	20	7	X \$18.00	\$ 126.00
Independent Claims	3	3	0	X \$78.00	\$.00
Multiple Dependent Claim(s), if any	0	0	0	X \$260.00	\$.00

☐ Amendment canceling extra claims enclosed

☐ Amendment deleting multiple dependencies enclosed

☐ Fee for extra claims is not being paid at this time

Filing Fee Calculation

\$ 886.00

9. Small Entity Statement

☐ verified statement that this is a filing by a small entity under 37 CFR 1.9 and 1.27 is attached.

Filing Fee Calculation (50% of above)

\$ _____

10. Fee Payment Being Made At This Time

☒ Enclosed

☒ basic filing fee

\$ 886.00

Total fees enclosed

\$ 886.00

11. Method of Payment of Fees

☒ check in the amount of \$ 886.00

12. Authorization to Charge Additional Fees

X The Commissioner is hereby authorized to charge the following additional fees which may be required to Account No. 18-1829;

X 37 CFR 1.16 (filing fees and presentation of extra claims)

X 37 CFR 1.17 (application processing fees)

 37 CFR 1.18 (issue fee at or before Mailing of Notice of Allowance, pursuant to 37 CFR 1.311(b)).

13. Instructions As To Overpayment

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PATENT

Replication-Competent Anti-Cancer Vectors

Reference to Government Grant

This invention was made with government support under a grant from the National Institutes of Health, Grant Number RO1 CA71704. The government has certain rights in this invention.

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Background of the Invention

(1) Field of the Invention

This invention relates generally to the treatment of cancer and more particularly to vectors which replicate in neoplastic cells and which overexpress an adenovirus death protein (ADP) and to the use of these vectors in treating human cancer.

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(2) Description of the Related Art

Cancer is a leading cause of death in the United States and elsewhere. Depending on the type of cancer, it is typically treated with surgery, chemotherapy, and/or radiation. These treatments often fail: surgery may not remove all the cancer; some cancers are resistant to chemotherapy and radiation therapy; and chemotherapy-resistant tumors frequently develop. New therapies are necessary, to be used alone or in combination with classical techniques.

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One potential therapy under active investigation is treating tumors with recombinant viral vectors expressing anti-cancer therapeutic proteins. Adenovirus-based vectors contain several characteristics that make them conceptually appealing for use in treating cancer, as well as for therapy of genetic disorders. Adenoviruses (hereinafter used interchangeably with "Ads") can easily be grown in culture to high titer stocks that are stable. They have a broad host range, replicating in most human cancer cell types. Their genome can be manipulated by site-directed mutation and insertion of foreign genes expressed from foreign promoters.

The adenovirion consists of a DNA-protein core within a protein capsid (reviewed by Stewart et al., "Adenovirus structure by x-ray crystallography and electron microscopy." in: *The Molecular Repertoire of Adenoviruses*, Doerfler, W. et al., (ed.), Springer-Verlag, Heidelberg, Germany, p. 25-38). Virions bind to a specific cellular receptor, are endocytosed, and the genome is extruded from endosomes and transported to the nucleus. The genome is a linear duplex DNA of about 36 kbp, encoding about 36 genes (Fig. 1A). In the nucleus, the "immediate early" E1A proteins are expressed initially, and these proteins induce expression of the "delayed early" proteins encoded by the E1B, E2, E3, and E4 transcription units (reviewed by Shenk, T. "Adenoviridae: the viruses and their replication" in: *Fields Virology*, Field, B.N. et al., Lippencott-Raven, Philadelphia, p. 2111-2148). E1A proteins also induce or repress cellular genes, resulting in stimulation of the cell cycle. About 23 early proteins function to usurp the cell and initiate viral DNA replication. Viral DNA replicates at about 7 h post-infection (p.i.), then late genes are expressed from the "major late" transcription unit. Major late mRNAs are synthesized from the common "major late promoter" by alternative pre-mRNA processing. Each late mRNA contains a common "tripartite leader" at its 5'-terminus (exons 1, 2, and 3 in Fig. 1), which allows for efficient translation of Ad late mRNAs. Cellular protein synthesis is shut off, and the cell becomes a factory for making viral proteins. Virions assemble in the nucleus at about 1 day p.i., and after 2-3 days the cell lyses and releases progeny virus. Cell lysis is mediated by the E3 11.6K protein, which has been renamed "adenovirus death protein" (ADP) (Tollefson et al., *J. Virol.* 70:2296-2306, 1996; Tollefson et al., *Virol.* 220:152-162, 1996). The term ADP as used herein in a generic sense refers collectively to ADP's from adenoviruses such as, e.g. Ad type 1 (Ad1), Ad type 2 (Ad2), Ad type 5 (Ad5) or Ad type 6 (Ad6) all of which express homologous ADP's with a high degree of sequence similarity.

The Ad vectors being investigated for use in anti-cancer and gene therapy are based on recombinant Ad's that are either replication-defective or replication-competent. Typical replication-defective Ad vectors lack the E1A and E1B genes (collectively known as E1) and contain in their place an expression cassette consisting of a promoter and pre-mRNA

processing signals which drive expression of a foreign gene. These vectors are unable to replicate because they lack the E1A genes required to induce Ad gene expression and DNA replication. In addition, the E3 genes are usually deleted because they are not essential for virus replication in cultured cells.

- 5 A number of investigators have constructed replication-defective Ad vectors expressing anti-cancer therapeutic proteins. Usually, these vectors have been tested by direct injection of human tumors growing in mouse models. Most commonly, these vectors express the thymidine kinase gene from herpes simplex virus, and the mice are treated with gancyclovir to kill cells transduced by the vector (see e.g., Felzmann et al., *Gene Ther.* 4:1322-1329, 1997). Another suicide gene therapy approach involves injecting tumors with a replication defective Ad vector expressing cytosine deaminase, followed by administration of 5-fluorocytosine (Topf et al., *Gene Ther.* 5:507-513, 1998). Investigators have also prepared and tested replication-defective Ad vectors expressing a cytokine-such as IL-2, IL-12, IL-6, tumor necrosis factor (TNF), type I interferons, or the co-stimulatory molecule B7-1 in the anticipation that the Ad-expressed cytokine will stimulate an immune response, including cytotoxic T-lymphocytes (CTL), against the tumor (Felzmann et al., *supra*; Putzer et al., *Proc. Natl. Acad. Sci. USA* 94:10889-10894, 1997). Other vectors express tumor antigens (e.g. melanoma MART1), proteins that de-regulate the cell cycle and induce apoptosis (p53, pRB, p21^{Kip1/WAF1}, p16^{CDKN2}, and even Ad E1A), and ribozymes. An Ad vector expressing FasL induces apoptosis and tumor regression of a mouse tumor (Arai et al., *Proc. Natl. Acad. Sci. USA* 94:13862-13867, 1997).

Despite these generally positive reports, it is recognized in the art that replication-defective Ad vectors have several characteristics that make them suboptimal for use in therapy. For example, production of replication-defective vectors requires that they be grown on a complementing cell line that provides the E1A proteins in trans. Such cell lines are fastidious, and generation of virus stocks is time-consuming and expensive. In addition, although many foreign proteins have been expressed from such vectors, the level of expression is low compared to Ad late proteins.

To address these problems, several groups have proposed using replication-competent Ad vectors for therapeutic use. Replication-competent vectors retain Ad genes essential for replication and thus do not require complementing cell lines to replicate. Replication-competent Ad vectors lyse cells as a natural part of the life cycle of the vector. Another advantage of replication-competent Ad vectors occurs when the vector is engineered to encode and express a foreign protein. Such vectors would be expected to greatly amplify synthesis of the encoded protein *in vivo* as the vector replicates. For use as anti-cancer agents,

replication-competent viral vectors would theoretically also be advantageous in that they should replicate and spread throughout the tumor, not just in the initial infected cells as is the case with replication-defective vectors.

Wyeth Laboratories developed replication-competent Ad vectors for vaccination purposes, using vaccine strains of Ad serotypes 4, 7, and 5 (Lubeck et al., *AIDS Res. Hum. Retroviruses* 10:1443-1449, 1994). Foreign genes were inserted into the E3 region (with the E3 genes deleted) or into a site at the right end of the genome. Two foreign genes used were hepatitis B surface antigen and the HIV envelope protein. They obtained good expression in culture, and were able to raise antisera in animal models. Phase I human trials were ambiguous, and the project was mostly abandoned.

Onyx Pharmaceuticals recently reported on adenovirus-based anti-cancer vectors which are replication deficient in non-neoplastic cells but which exhibit a replication phenotype in neoplastic cells lacking functional p53 and/or retinoblastoma (pRB) tumor suppressor proteins (U.S. Patent No. 5,677,178; Heise et al., *Nature Med.* 6:639-645, 1997; Bischoff et al., *Science* 274:373-376, 1996). This phenotype is reportedly accomplished by using recombinant adenoviruses containing a mutation in the E1B region that make the encoded E1B-55K protein incapable of binding to p53 and/or a mutation(s) in the E1A region which make the encoded E1A protein (p289R or p243R) incapable of binding to pRB and/or the cellular 300 kD polypeptide and/or the 107 kD polypeptide. E1B-55K has at least two independent functions: it binds and inactivates the tumor suppressor protein p53, and it is required for efficient transport of Ad mRNA from the nucleus. Because these E1B and E1A viral proteins are involved in forcing cells into S-phase, which is required for replication of adenovirus DNA, and because the p53 and pRB proteins block cell cycle progression, the recombinant adenovirus vectors described by Onyx should replicate in cells defective in p53 and/or pRB, which is the case for many cancer cells, but not in cells with wild-type p53 and/or pRB. Onyx has reported that replication of an adenovirus lacking E1B-55K, which is named ONYX-015, was restricted to p53-minus cancer cell lines (Bischoff et al., *supra*), and that ONYX-015 slowed the growth or caused regression of a p53-minus human tumor growing in nude mice (Heise et al., *supra*). Others have challenged the Onyx report claiming that replication of ONYX-015 is independent of p53 genotype and occurs efficiently in some primary cultured human cells (Harada and Berk, *J. Virol* 73:5333-5344, 1999). ONYX-015 does not replicate as well as wild-type adenovirus because E1B-55K is not available to facilitate viral mRNA transport from the nucleus. Also, ONYX-015 expresses less ADP than wild-type virus (see Example 1 below).

As an extension of the ONYX-015 concept, a replication-competent adenovirus vector was designed that has the gene for E1B-55K replaced with the herpes simplex virus thymidine kinase gene (Wilder et al., *Gene Therapy* 6:57-62, 1999). The group that constructed this vector reported that the combination of the vector plus gancyclovir showed a therapeutic effect on a human colon cancer in a nude mouse model (Wilder et al., *Cancer Res.* 59:410-413, 1999). However, this vector lacks the gene for ADP, and accordingly, the vector will lyse cells and spread from cell-to-cell less efficiently than an equivalent vector that expresses ADP. The gene for ADP is also lacking in another replication-competent adenovirus vector that has been described, in which a minimal enhancer/promoter of the human prostate specific antigen was inserted into the adenovirus E1A enhancer/promoter (Rodriguez et al., *Cancer Res.* 57:2559-2563, 1997).

Thus, there is a continuing need for vectors that replicate and spread efficiently in tumors but that can be modified such that they replicate poorly or not at all in normal tissue.

15 Summary of the Invention

Briefly, therefore, the present invention is directed to novel vectors which are replication competent in neoplastic cells and which overexpress an adenovirus death protein (ADP). The work reported herein demonstrates the discovery that overexpression of ADP by a recombinant adenovirus allows the construction of a replication-competent adenovirus that kills neoplastic cells and spreads from cell-to-cell at a rate similar to or faster than that exhibited by adenoviruses expressing wild-type levels of ADP, even when the recombinant adenovirus contains a mutation that would otherwise reduce its replication rate in non-neoplastic cells. This discovery was unexpected because it could not have been predicted from what was known about adenovirus biology that Ad vectors overexpressing ADP remain viable and that the infected cells are not killed by the higher amounts of ADP before the Ad vector produces new virus particles that can spread to other tumor cells. Indeed, naturally-occurring adenoviruses express ADP in low amounts from the E3 promoter at early stages of infection, and begin to make ADP in large amounts only at 24-30 h p.i., once virions have been assembled in the cell nucleus. It is believed that other non-adenoviral vectors can be used to deliver ADP's cell-killing activity to neoplastic cells, including other viral vectors and plasmid expression vectors.

Thus, in one preferred embodiment, the ADP-expressing vector comprises a recombinant adenovirus lacking expression of at least one E3 protein selected from the group consisting of: gp19K; RID α (also known as 10.4K); RID β (also known as 14.5K) and 14.7K. Because these E3 proteins inhibit immune-mediated inflammation and/or apoptosis of Ad-

infected cells, it is believed that a recombinant adenovirus lacking one or more of these E3 proteins will stimulate infiltration of inflammatory and immune cells into a tumor treated with the adenovirus and that this host immune response will aid in destruction of the tumor as well as tumors that have metastasized. The ADP expressed by preferred embodiments comprises a naturally-occurring amino acid sequence from a human adenovirus of subgroup C, namely Ad1, Ad2, Ad5 and Ad6.

In another embodiment, replication of the vector is restricted to neoplastic cells. Such replication-restricted vectors are useful in treating cancer patients in which it is desirable to eliminate or reduce damage to normal cells and tissues that might be caused by the vector, particularly viral vectors that kill the host cell as part of their life cycle. In preferred embodiments, a recombinant adenovirus has a replication-restricted phenotype because the recombinant adenovirus is incapable of expressing an E1A viral protein which binds the pRB and the p300/CBP proteins or because the E4 promoter has been substituted with a promoter that is activated only in neoplastic cells.

In yet another embodiment, the invention provides a vector which overexpresses ADP and whose replication is under the control of a tissue specific promoter or an inducible promoter. In preferred embodiments, the vector comprises a recombinant adenovirus in which the tissue specific promoter or inducible promoter is substituted for the E4 promoter. Such vectors are useful for restricting replication of the vector and its ADP-mediated cell killing to cells of a particular type or to cells exposed to an exogenous agent that activates the promoter. A preferred tissue-specific or inducible vector also expresses a phenotype that restricts its replication to neoplastic cells.

In yet another embodiment, the invention provides a vector which overexpresses ADP but which is not restricted to tumors by a specific genetic modification. Such a vector is more destructive to neoplastic cells than even the naturally occurring Ad's of subgroup C. In preferred embodiments, this vector could be used for patients with terminal cancer not treatable by another method, and who have pre-existing neutralizing antibodies to Ad or to which neutralizing antibodies can be administered.

In still another embodiment, the invention provides a composition comprising a first recombinant virus which is replication competent in a neoplastic cell and overexpresses the adenovirus death protein. In one embodiment, the recombinant virus is contained within a delivery vehicle comprising a targeting moiety that limits delivery of the virus to cells of a certain type. With this embodiment, the replication-competent vector can be of any ADP-overexpressing configuration described herein. In some embodiments, the composition also comprises a second recombinant virus which is replication-defective and which expresses an

anti-cancer gene product. The recombinant virus complements spread of the replication-defective virus, as well as its encoded anti-cancer product, throughout a tumor. In preferred embodiments, the first recombinant virus is a recombinant adenovirus whose replication is restricted to neoplastic cells and/or which lacks expression of one or more of the E3 gp19K;

5 RID α ; RID β ; and 14.7K proteins.

The ADP-expressing vectors and compositions of the invention are useful in a method for promoting death of a neoplastic cell. The method comprises contacting the neoplastic cell with a vector which is replication-competent in the neoplastic cell and which overexpresses ADP. Where the neoplastic cell comprises a tumor in a patient, the vector is
10 administered directly to the tumor or, in other embodiments, the vector is administered to the patient systemically or in a delivery vehicle containing a targeting moiety that directs delivery of the vector to the tumor. In embodiments where the vector is a recombinant virus, the method can also comprise passively immunizing the patient against the virus.

In yet another embodiment of the invention, the vector may be used in combination
15 with radiation therapy. The radiation therapy can be any form of radiation therapy used in the art such as for example, external beam radiation such as x-ray treatment, radiation delivered by insertion of radioactive materials within the body near or at the tumor site such as treatment with gamma ray emitting radionuclides, particle beam therapy which utilizes neutrons or charged particles and the like. In addition, this embodiment encompasses the use
20 of more than one of the vectors of the present invention in a cocktail in combination with radiation therapy.

Another embodiment of the invention involves the use of the recombinant vector in combination with chemotherapy as has been disclosed for other adenovirus vectors (U.S. Patent No. 5,846,945). Chemotherapeutic agents are known in the art and include
25 antimetabolites including pyrimidine-analogue and purine-analogue antimetabolites, plant alkaloids, antitumor antibiotics, alkylating agents and the like. The use of more than one of the vectors of the present invention with a chemotherapeutic agent or agents is also contemplated within this embodiment.

Among the several advantages found to be achieved by the present invention,
30 therefore, may be noted the provision of replication-competent vectors, particularly viruses, which rapidly kill cancer cells and spread from cell-to-cell in a tumor; the provision of such vectors whose replication can be induced or which is restricted to tumors and/or to cells of a certain tissue type; and the provision of compositions and methods for anti-cancer therapy which cause little to no side effects in normal tissues.

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Brief Description of the Drawings

Figure 1 is a schematic of gene expression in Ad5 (Fig. 1A) and KD3, a preferred embodiment of the invention (Fig. 1B), in which the respective genomes are represented by the stippled bars and transcription units represented by arrows above and below the bars, with the E3 proteins listed above the arrows for the E3 transcription unit, and the L1 to L5 families of late mRNA's indicated.

Figure 2 illustrates the overexpression of ADP by KD1, KD3, GZ1, and GZ3 showing an immunoblot of proteins isolated from human A549 cells infected with the indicated viruses and probed with an anti-ADP antibody, with ADP indicating differently glycosylated and proteolytically processed forms of ADP.

Figure 3 illustrates that the E1A *dl1101/1107* mutation referred to in the figure and hereinafter as *dl01/07*, retards expression of late proteins, showing an immunoblot of E1A proteins and late proteins in A549 cells infected with the indicated viruses in the absence (Figs. 3A and 3B) or presence (Figs. 3C and 3D) of *dl327*, which has a wild-type E1A region and has a deletion of all E3 genes but the gene encoding the 12.5K protein (Figs. 3C and 3D). An antiserum specific to the E1A proteins was used for Fig. 3A and 3C. An antiserum raised against Ad5 virions was used for Figs. 3B and 3D.

Figure 4 illustrates that KD1 and KD3 kill cells more efficiently than control viruses that express less or no ADP, showing a graph of the percent of A549 cells infected with the indicated viruses that were viable at the indicated days p.i. as determined by trypan blue exclusion.

Figure 5 is a cell spread assay illustrating that overexpression of ADP enhances spread of virus from cell to cell, showing monolayers infected with the indicated viruses at the indicated PFU/cell which were treated at 7 days p.i. with crystal violet, which stains live cells but not dead cells.

Figure 6 illustrates that KD1 and KD3 replicate well in growing cells but not in growth-arrested cells showing the virus titer extracted from growing or growth arrested HEL-229 cells at various times following infection with 100 PFU/ml of the following viruses: *dl309* (Fig. 6A), *dl01/07* (fig. 6B), KD1 (Fig. 6C) and KD3 (Fig 6D).

Figure 7 illustrates that KD1 and KD3 are defective in killing primary human bronchial epithelial cells showing these cell monolayers infected at 30% confluency with 10 PFU/ml of the indicated viruses and stained at 5 days p.i. with neutral red.

Figure 8 illustrates that KD1 and KD3 reduce the growth rate of human A549 cell tumors growing in nude mice, showing in Fig. 8A a graph of average-fold increase in tumor size plotted against the number of weeks following infection of the tumor with buffer or with

5 x 10⁷ PFU at weekly intervals of or the indicated viruses, and showing in Fig. 8B a similar graph of tumors injected once with 5 x 10⁸ PFU of KD3 or GZ3.

Figure 9 illustrates that KD1 and KD3 reduce the growth rate of human Hep3B cell tumors growing in nude mice, showing a graph of average-fold increase in tumor size plotted against the number of weeks following injection of the tumor with buffer or with 5 x 10⁷ PFU of *dI309*, KD1 or KD3 at twice weekly intervals of the indicated viruses.

Figure 10 illustrates that KD1 and KD3 complement the replication and spread of Ad- β -gal, a replication-defective vector that expresses β -galactosidase, using an infectious center assay showing in Fig. 10A a picture of A549 cell monolayers seeded with A549 cells infected with Ad- β -gal alone or with the indicated viruses, with Figs 10B and 10C showing close-up views of two of the monolayers of Fig. 10A.

Figure 11 is a bar graph illustrating that KD1 and KD3 increase the expression of luciferase in human Hep3B cell tumors growing in nude mice, using an assay in which tumors were injected with the indicated combinations of viruses, then were extracted 2 weeks p.i. and assayed for luciferase activity. The numbers in parentheses indicated the fold increase in luciferase activity compared to that of the Adluc vector plus buffer.

Figure 12 is a graph showing the results of a standard plaque development assay for KD1 and KD1-SPB on A549 cells engineered to express the TTF1 transcription factor (A549/TTF1) and the parental 549 cells, in which data are plotted as the number of plaques observed on a particular day in the assay divided by the final number of plaques observed for that virus multiplied by 100.

Figure 13 is a cell spread assay for KD1 and KD1-SPB on H441 cells and Hep3B cells, where cells were infected with the indicated amounts of KD1 or KD1-SPB and H441 cells and Hep3B cells were stained with crystal violet at 5 days p.i. and 8 days p.i., respectively.

Figure 14 is a graph showing the results of a standard plaque development assay for *dI309* and two preferred embodiments of the invention, GZ1 and GZ3, in which data are plotted as the number of plaques observed on a particular day in the assay divided by the final number of plaques observed for that virus multiplied by 100.

Figure 15 is a cell spread assay illustrating that the combination of KD1, KD3, GZ1, or GZ3 with x-ray radiation is more effective in destroying A549 cell monolayers than is virus vector alone or radiation alone, wherein cells were infected with the indicated amounts of the indicated viruses, radiated with 600 centigrays (cGy) of x-radiation (bottom panel), or mock radiated (top panel), then stained with crystal violet at 6 days p.i.

Figure 16 is a graph of a cell spread assay illustrating that 10^{-3} PFU of KD1, KD3, GZ1, or GZ3 used in combination with 150, 300, or 600 centigreys of radiation is more effective in destroying A549 cell monolayers than virus vector alone or radiation alone. Cell viability is based on the amount of crystal violet extracted from the culture wells, using the mock-infected non-radiated well as 100% viability.

Figure 17 illustrates that the combination of KD3 or GZ3 plus x-ray radiation is more effective in reducing the growth of A549 cell tumors growing in nude mice than KD3 alone or GZ3 alone.

Figure 18 illustrates a structure-function analysis of ADP, showing in Fig. 18A the amino acid sequence of the adenovirus death protein encoded by Ad2, with the various putative domains and glycosylation sites labeled and showing in Fig. 18B a schematic of the ADP gene in *rec700* and in the indicated deletion mutants, with the right column summarizing the death promoting phenotype of the various mutants as a percentage of the wild-type phenotype.

Figures 19A and 19B illustrate a cell viability assay of the indicated ADP mutants showing a graph of viability as determined by trypan blue exclusion plotted against hours (Fig. 19A) or days (Fig. 19B) postinfection.

Figure 20 depicts the amino acid sequence, shown in single letter code, for the ADP proteins of Ad1, Ad2, Ad5, and Ad6 (SEQ ID NOS:5-8), for the Ad2 ADP mutants *dl716*, *dl715*, *dl714*, and *dl737* (SEQ ID NOS:9-12), and for putative luminal Domain (SEQ ID NO:17), transmembrane domain (SEQ ID NO:18), the cytosolic basic-proline domain (SEQ ID NO:19), and the remainder of the cytosolic domain (SEQ ID NO:20) of the ADP protein of Ad2.

Figure 21 presents the complete nucleotide sequence of the genome of Ad5.

Figure 22 presents the complete nucleotide sequence of the genome of KD1 (SEQ ID NO:1).

Figure 23 presents the complete nucleotide sequence of the genome of KD3 (SEQ ID NO:2).

Description of the Preferred Embodiments

In accordance with the present invention, it has been discovered that overexpression of ADP by a recombinant adenovirus results in faster lysis of cells and spread of the virus throughout a cell monolayer than viruses expressing wild-type levels of ADP. It has also been discovered that this function for ADP is manifest in an adenovirus which contains E1A mutations that restrict adenoviral replication to neoplastic cells. Thus, vectors which are both

replication competent in neoplastic cells and which overexpress ADP should be useful in anti-cancer therapy.

In the context of this disclosure, the following terms will be defined as follows unless otherwise indicated:

5 "Naturally-occurring" as applied to an object such as a polynucleotide, polypeptide, or virus means that the object can be isolated from a source in nature and has not been intentionally modified by a human.

"Neoplastic cell" means a cell which exhibits an aberrant growth phenotype characterized by a significant loss of control of cell proliferation and includes actively
10 replicating cells as well as cells in a temporary non-replicative resting state (G_1 or G_2). A neoplastic cell may have a well-differentiated phenotype or a poorly-differentiated phenotype and may comprise a benign neoplasm or a malignant neoplasm.

"Recombinant virus" means any viral genome or virion which is different than a wild-type virus due to a deletion, insertion, or substitution of one or more nucleotides in the wild-
15 type viral genome. The recombinant virus can have changes in the number of amino acid sequences encoded and expressed or in the amount or activity of proteins expressed by the virus. In particular, the term includes recombinant viruses generated by the intervention of a human.

"Replication-competent" as applied to a vector means that the vector is capable of
20 replicating in normal and/or neoplastic cells. As applied to a recombinant virus, "replication-competent" means that the virus exhibits the following phenotypic characteristics in normal and/or neoplastic cells: cell infection; replication of the viral genome; and production and release of new virus particles; although one or more of these characteristics need not occur at the same rate as they occur in the same cell type infected by a wild-type virus, and may occur
25 at a faster or slower rate. Where the recombinant virus is derived from a virus such as adenovirus that lyses the cell as part of its life cycle, it is preferred that at least 5 to 25% of the cells in a cell culture monolayer are dead 5 days after infection. Preferably, a replication-competent virus infects and lyses at least 25 to 50%, more preferably at least 75%, and most preferably at least 90% of the cells of the monolayer by 5 days post infection (p.i.).

30 "Replication-defective" as applied to a recombinant virus means the virus is incapable of or is greatly compromised in, replicating its genome in any cell type in the absence of a complementing replication-competent virus. Exceptions to this are cell lines such as 293 cells that have been engineered to express adenovirus E1A and E1B proteins.

"Replication-restricted" as applied to a vector of the invention means the vector
35 replicates better in a dividing cell, i.e. either a neoplastic cell or a non-neoplastic, dividing

cell, than in a cell of the same type that is not neoplastic and/or not dividing, which is also referenced herein as a normal, non-dividing cell. Preferably, a replication-restricted virus kills at least 10% more neoplastic cells than normal, non-dividing cells in cell culture monolayers of the same size, as measured by the number of cells showing cytopathic effects (CPE) at 5 days p.i. More preferably, between 25% and 50%, and even more preferably, between 50% and 75% more neoplastic than normal cells are killed by a replication-restricted virus. Most preferably, a replication-restricted adenovirus kills between 75% and 100% more neoplastic than normal cells in equal sized monolayers by 5 days p.i.

In one embodiment the invention provides a vector that is replication-competent in neoplastic cells and which overexpresses an ADP. Vectors useful in the invention include but are not limited to plasmid-expression vectors, bacterial vectors such as *Salmonella* species that are able to invade and survive in a number of different cell types, vectors derived from DNA viruses such as human and non-human adenoviruses, adenovirus associated viruses (AAVs), poxviruses, herpesviruses, and vectors derived from RNA viruses such as retroviruses and alphaviruses. Preferred vectors include recombinant viruses engineered to overexpress an ADP. Recombinant adenoviruses are particularly preferred for use as the vector, especially vectors derived from Ad1, Ad2, Ad5 or Ad6.

Vectors according to the invention overexpress ADP. As applied to recombinant Ad and AAV vectors, the term "overexpresses ADP" means that more ADP molecules are made per viral genome present in a dividing cell infected by the vector than expressed by any previously known recombinant adenoviral vector or AAV in a dividing cell of the same type. As applied to other, non-adenoviral vectors, "overexpresses ADP" means that the virus expresses sufficient ADP to lyse a cell containing the vector.

Vectors overexpressing ADP can be prepared using routine methodology. (See, e.g., *A Laboratory Cloning Manual*, 2nd Ed., vol. 3, Sambrook et al., eds., Cold Spring Harbor Laboratory Press, 1989). For example, a polynucleotide encoding the ADP can be cloned into a plasmid expression vector known to efficiently express heterologous proteins in mammalian cells. The polynucleotide should also include appropriate termination and polyadenylation signals. Enhancer elements may also be added to the plasmid to increase the amount of ADP expression. Viral vectors overexpressing ADP can be prepared using similar materials and techniques.

Where the virus is a recombinant adenovirus, overexpression of ADP can be achieved in a multitude of ways. In general, any type of deletion in the E3 region that removes a splice site for any of the E3 mRNAs will lead to overexpression of the mRNA for ADP, inasmuch as more of the E3 pre-mRNA molecules will be processed into the mRNA for ADP. This is

exemplified in the KD1, KD3, GZ1 and GZ3 vectors (SEQ ID NOS:1-4) whose construction is described below. Other means of achieving overexpression of ADP in Ad vectors include, but are not limited to: insertion of pre-mRNA splicing and cleavage/polyadenylation signals at sites flanking the gene for ADP; expression of ADP from another promoter, e.g. the human cytomegalovirus promoter, inserted into a variety of sites in the Ad genome; and insertion of the gene for ADP behind the gene for another Ad mRNA, together with a sequence on the 5' side of the ADP sequence that allows for internal initiation of translation of ADP, e.g. the Ad tripartite leader or a viral internal ribosome initiation sequence.

The ADP expressed by a vector according to the invention is any polypeptide comprising a naturally-occurring full-length ADP amino acid sequence or variant thereof that confers upon a vector expressing the ADP the ability to lyse a cell containing the vector such that replicated copies of the vector are released from the infected cell. A preferred full-length ADP comprises the ADP amino acid sequence encoded by Ad1, Ad2, Ad5 or Ad6. These naturally-occurring ADP sequences are set forth in SEQ ID NOS:5-8, respectively. ADP variants include fragments and deletion mutants of naturally-occurring adenovirus death proteins, as well as full-length molecules, fragments and deletion mutants containing conservative amino acid substitutions, provided that such variants retain the ability, when expressed by a vector inside a cell, to lyse the cell.

Conservative amino acid substitutions refer to the interchangeability of residues having similar side chains. Conservatively substituted amino acids can be grouped according to the chemical properties of their side chains. For example, one grouping of amino acids includes those amino acids having neutral and hydrophobic side chains (A, V, L, I, P, W, F, and M); another grouping is those amino acids having neutral and polar side chains (G, S, T, Y, C, N, and Q); another grouping is those amino acids having basic side chains (K, R, and H); another grouping is those amino acids having acidic side chains (D and E); another grouping is those amino acids having aliphatic side chains (G, A, V, L, and I); another grouping is those amino acids having aliphatic-hydroxyl side chains (S and T); another grouping is those amino acids having amine-containing side chains (N, Q, K, R, and H); another grouping is those amino acids having aromatic side chains (F, Y, and W); and another grouping is those amino acids having sulfur-containing side chains (C and M). Preferred conservative amino acid substitutions groups are: R-K; E-D, Y-F, L-M; V-I, and Q-H.

As used herein, an ADP variant can also include modifications of a naturally-occurring ADP in which one or more amino acids have been inserted, deleted or replaced with a different amino acid or a modified or unusual amino acid, as well as modifications such as

glycosylation or phosphorylation of one or more amino acids so long as the ADP variant containing the modified sequence retains cell lysing activity.

As described below, the inventors herein performed a structure-function analysis of ADP which defined specific domains in ADP required to promote cell death. Using this information, when combined with known recombinant DNA and cloning methodology, it is believed the skilled artisan can readily construct ADP variants of a naturally-occurring adenovirus death protein and test them for cell lysing activity. A preferred ADP deletion mutant comprises an ADP amino acid sequence from any of the deletion mutants *dl716*, *dl715*, *dl714* and *dl737*, whose ADP sequences are set forth in SEQ ID NOS:9-12, respectively).

Where the vector is derived from a virus, it is preferred that the virus lack expression of one or more viral proteins involved in avoiding host anti-viral defenses such as immune-mediated inflammation and/or apoptosis of infected cells. For example, adenovirus contains a cassette of genes that prevents killing of Ad-infected cells by the immune system (Wold et al., *Semin. Virol.*, 1998 (8:515-523, 1998). The E3-14.7K protein and the E3 RID (Receptor Internalization and Degradation) protein, which is a complex consisting of RID α and RID β , inhibit apoptosis of Ad-infected cells induced by tumor necrosis factor (TNF) and the Fas ligand which are expressed on, or secreted by, activated macrophages, natural killer (NK) cells, and cytotoxic lymphocytes (CTLs) (Tollefson et al., *Nature* 392:727-730, 1998). The E3-gp19K protein inhibits CTL-killing of infected cells by blocking transport of MHC class I antigens to the cell surface (Wold et al., *supra*). Thus, it is believed that infection of tumor cells by such viral vectors will stimulate infiltration of inflammatory cells and lymphocytes into the tumor, and will not prevent infected tumor cells from apoptosis induced by cytolytic cells of the immune system, or against apoptosis inducing cytokines. For example, it is known that when mice are infected with Ad mutants lacking the E3 gp19K, RID and 14.7K proteins there is a dramatic increase (as compared to E3-positive Ad) in infiltration of inflammatory cells and lymphocytes into the infected tissue (Sparer et al., *J. Virol.* 70:2431-2439, 1996). A similar infiltration of tumors infected by an ADP-expressing viral vector of the invention would be expected to further promote destruction of the tumor by adding an immune system attack to the ADP-mediated killing activity. For example, it is believed that the viral infection will stimulate formation of tumor-specific CTL's that can kill neoplastic cells not only in the tumor but also ones that have metastasized. In addition, it is also expected that vector-specific CTL's will be generated which could attack vector-infected cells if the vector spreads away from the tumor into normal cells. Because viral vectors

overexpressing ADP will spread rapidly through the tumor, it is believed these immune mechanisms will have little effect on spread of the vector.

Where the vector is a recombinant adenovirus, it is preferred that the adenovirus lack expression of each of the E3 gp19K, RID, and 14.7K proteins. By "lack expression" and "lacking expression" of a protein(s), "it is meant" that the viral genome contains one or more mutations that inactivates expression of a functional protein, i.e., one having all the functions of the wild-type protein. The inactivating mutation includes but is not limited to substitution or deletion of one or more nucleotides in the encoding gene(s) that prevents expression of functional transcripts or that results in transcripts encoding nonfunctional translation products.

A particularly preferred way to inactivate expression of the Ad E3 gp19K, RID, and 14.7K proteins is by deleting the E3 region containing the genes encoding these proteins. Preferably, one or both of the E3 genes encoding the E3 6.7K and 12.5K proteins are also deleted because, as discussed in the Examples below, it is believed that deletion of most or all of the E3 genes other than the ADP gene facilitates overexpression of ADP mRNA by reducing competition for splicing of the major late pre-mRNAs. Preferred Ad vectors containing an E3 deletion that overexpress ADP are GZ1 (SEQ ID NO:3) and GZ3 (SEQ ID NO:4), whose construction and properties are described in the Examples below.

The invention also provides ADP-expressing vectors whose replication is restricted to dividing cells. Any means known to provide such a replication-restricted phenotype may be used. For example, WO 96/40238 describes microbes that preferentially invade tumor cells as well as methods for identifying and isolating bacterial promoters that are selectively activated in tumors. It is also contemplated that expression of one or more vector proteins essential for replication can be placed under the control of the promoter for a cellular gene whose expression is known to be upregulated in neoplastic cells. Examples of such genes include but are not limited to: the breast cancer markers mammaglobin (Watson et al., *Oncogene* 16:817-824, 1998); BRCA1 (Norris et al., *J. Biol. Chem.* 270:22777-22782, 1995) and *her2/neu* (Scott et al., *J. Biol. Chem.* 269:19848-19858, 1994); and prostate specific antigen (U.S. Patent 5,698,443); surfactant protein B for lung alveoli (Yan et al., *J. Biol. Chem.* 270:24852-24857, 1995); factor VII for liver (Greenberg et al., *Proc. Natl. Acad. Sci. USA* 92:12347-12351, 1995); and survivin for cancer in general (Li et al., *Nature* 396:580-584). Where the vector is an adenovirus, it is contemplated that such tumor-specific promoters can be substituted for the E4 promoter. Because E4 gene products are essential for Ad replication, placing their expression under the control of a tumor-specific promoter should restrict replication of the vector to tumor cells in which the promoter is activated.

Another strategy for restricting replication of ADP-expressing Ad vectors to neoplastic cells is exemplified by the KD1 (SEQ ID NO:1), KD2 (SEQ ID NO:13) and KD3 (SEQ ID NO:2) vectors, whose construction and properties are described in the Examples below. This strategy exploits a pre-existing Ad5 mutant in the E1A gene, named *dl1101/1107* (Howe et al., *Proc. Natl. Acad. Sci.*, 87:5883-5887, 1990), also referred to herein as *dl01/07*, and which can only grow well in cancer cells. The role of E1A is to drive cells from the G₀ and G₁ phases of the cell cycle into S-phase. This is achieved by two mechanisms, one involving pRB (and family members), and the other involving p300 and the related protein CBP (DePinho, R.A., *Nature* 391:533-536, 1998). One domain in E1A binds members of the pRB family. pRB normally exists in the cell as a complex with the transcription factor E2F-1 and E2F family members (E2F), tethered via E2F to E2F binding sites in promoters of cells expressed in S-phase. Here, pRB acts as a transcriptional co-repressor. E1A binding to pRB relieves this repression, and causes the release of E2F from pRB/E2F complexes. Free E2F then activates promoters of genes expressed in S-phase, e.g. thymidine kinase, ribonucleotide reductase, etc. Another domain in E1A binds the p300/CBP transcription adaptor protein complex. p300/CBP is a transcriptional co-activator that binds many different transcription factors and accordingly is targeted to promoters. p300/CBP has intrinsic histone acetyltransferase activity. E1A binding to p300/CBP is believed to inhibit this histone acetyltransferase activity, allowing acetylation of histones and repression of transcription (Chakravarti et al., *Cell* 96:393-403, 1999; Hamamori et al., *Cell* 96:405-413, 1999). Conceivably, some of the genes that are repressed as a result of E1A interacting with p300/CBP to play a role in blocking the cell cycle, although this is not known. Cancer cells are cycling, so they have free E2F and presumably some p300/CBP-regulated genes are repressed. Consistent with these ideas, E1A must bind both p300/CBP and the pRB family in order to transform primary cells to a constitutively cycling state (Howe et al., *supra*). The mutant *dl01/07* lacks both the p300/CBP- and pRB-binding domains and, as expected, it replicates very poorly in non-dividing "normal" cells or serum-starved cancer cells, but well in growing cancer cells. As described below, the growth of the KD1 and KD3 vectors, which contain the *dl01/07* E1A mutation, is very much better in dividing cancer cells as compared to non-dividing cells. Because the *dl01/07* mutant is completely defective in oncogenic transformation of rat cells (Howe et al., *supra*), vectors according to the invention that contain this E1A mutation cannot induce cancer in humans (remote as that may be) through an E1A-dependent mechanism.

The invention also includes vectors overexpressing ADP whose replication is restricted to specific tissues by placing expression of one or more proteins essential for

replication under the control of a tissue specific promoter. A number of tissue-specific promoters have been described in the art such as the surfactant protein B promoter which is only active in cells containing the TTF1 transcription factor, i.e., type II alveolar cells (Yan et al., *supra*) the transcriptional regulatory element described in U.S. Patent 5,466,596 to Breitman et al., that directs gene expression specifically in cells of endothelial lineage; prostate specific antigen which is expressed in prostate cells (Rodriguez et al., *supra*); and human alpha-lactalbum gene which is expressed in breast cancer cells (Anderson et al., *Gene Therapy* 6:854-864, 1999). Many other tissue-specific or tissue-preferred enhancer/promoters have been reported (Miller and Whelan, *Human Gene Therapy* 8:803-815, 1997).

Replication of vectors according to the invention can also be controlled by placing one or more genes essential for vector replication under the control of a promoter that is activated by an exogenous inducing agent, such as metals, hormones, antibiotics, and temperature changes. Examples of such inducible promoters include but are not limited to metallothionein promoters, the glucocorticoid promoter, the tetracycline response promoter, and heat shock protein (hsp) promoters such as the hsp 65 and 70 promoters.

The invention also provides compositions comprising a recombinant vector that overexpresses ADP in an amount effective for promoting death of neoplastic cells and a method comprising administering a therapeutically effective amount of the vector to a neoplastic cell in a patient. It is believed the compositions and methods of the present invention are useful for killing neoplastic cells of any origin and include neoplastic cells comprising tumors as well as metastatic neoplastic cells.

It is also contemplated that ADP-expressing viral vectors can be administered to neoplastic cells along with a replication-defective virus that expresses an anti-cancer gene product. For example, many replication-defective E1⁻ Ad vectors for use in cancer therapy are well characterized. A limitation of replication-defective vectors is that they only synthesize the therapeutic protein in the cell they initially infect, they cannot spread to other cells. Also, since the genome does not replicate, transcription can only occur from the input genomes, and this could be as low as one copy per cell. In contrast, the genome of replication-competent Ad vectors are amplified by about 10^4 in the cell that was initially infected, providing more templates for transcription. More amplification is achieved as the vector spreads to other cells. By combining replication-defective viral vectors expressing an anti-cancer gene product with replication-competent viral vectors described herein, it is expected that the result will be template amplification and rapid spread of both vectors to surrounding cells. For example, with Ad-based vectors, the burst size for each vector should be large, $\sim 10^4$ PFU/cell, so the probability of co-infection of surrounding cells by both vectors

will be high. Thus, both the replication-competent and replication-defective vectors should spread simultaneously through the tumor, providing even more effective anti-cancer therapy.

Expression of the anti-cancer gene product encoded by the replication-defective vector can be under the control of either constitutive, inducible or cell-type specific promoters. The anti-cancer gene product can be any substance that promotes death of a neoplastic cell. The term "gene product" as used herein refers to any biological product or products produced as a result of the biochemical reactions that occur under the control of a gene. The gene product can be, for example, an RNA molecule, a peptide, a protein, or a product produced under the control of an enzyme or other molecule that is the initial product of the gene, i.e., a metabolic product. For example, a gene can first control the synthesis of an RNA molecule which is translated by the action of ribosomes into a prodrug converting enzyme which converts a nontoxic prodrug administered to a cancer patient to a cell-killing agent; the RNA molecule, enzyme, and the cell-killing agent generated by the enzyme are all gene products as the term is used here. Examples of anti-cancer gene products include but are not limited to cell-killing agents such as apoptosis-promoting agents and toxins; prodrug converting enzymes; angiogenesis inhibitors; and immunoregulatory molecules and antigens capable of stimulating an immune response, humoral and/or cellular, against the neoplastic cell.

Apoptosis-promoting agents include but are not limited to the pro-apoptotic members of the BCL-2 family such as BAX, BAD, BID and BIK, as well as antisense molecules which block expression of anti-apoptotic members of the BCL-2 family. Examples of immunoregulatory molecules are cytokines such as tumor necrosis factor, Fas/Apo1/CD95 ligand, tumor necrosis factor related apoptosis inducing ligand, interleukins, macrophage activating factor and interferon γ . Angiogenesis inhibitors include but are not limited to endostatin and angiostatin. Toxins include but are not limited to tumor necrosis factor, lymphotoxin, the plant toxin ricin, which is not toxic to humans due to the lack of ricin receptors in animal cells, and the toxic subunit of bacterial toxins. Examples of pro-drug converting enzymes and pro-drug combinations are described in WO 96/40238 and include: thymidine kinase and acyclovir or gancyclovir; and bacterial cytosine deaminase and 5-fluorocytosine.

The therapeutic or pharmaceutical compositions of the present invention can be administered by any suitable route known in the art including for example by direct injection into a tumor or by other injection routes such as intravenous, subcutaneous, intramuscular, transdermal, intrathecal and intracerebral. Administration can be either rapid as by injection or over a period of time as by slow infusion or administration of slow release formulation.

For treating tissues in the central nervous system, administration can be by injection or infusion into the cerebrospinal fluid (CSF). When it is intended that a recombinant vector of the invention be administered to cells in the central nervous system, administration can be with one or more agents capable of promoting penetration of the vector across the blood-brain barrier. Preferably, vectors of the invention are administered with a carrier such as liposomes or polymers containing a targeting moiety to limit delivery of the vector to targeted cells. Examples of targeting moieties include but are not limited to antibodies, ligands or receptors to specific cell surface molecules.

Compositions according to the invention can be employed in the form of pharmaceutical preparations. Such preparations are made in a manner well known in the pharmaceutical art. One preferred preparation utilizes a vehicle of physiological saline solution, but it is contemplated that other pharmaceutically acceptable carriers such as physiological concentrations of other non-toxic salts, five percent aqueous glucose solution, sterile water or the like may also be used. It may also be desirable that a suitable buffer be present in the composition. Such solutions can, if desired, be lyophilized and stored in a sterile ampoule ready for reconstitution by the addition of sterile water for ready injection. The primary solvent can be aqueous or alternatively non-aqueous.

The carrier can also contain other pharmaceutically-acceptable excipients for modifying or maintaining the pH, osmolarity, viscosity, clarity, color, sterility, stability, rate of dissolution, or odor of the formulation. Similarly, the carrier may contain still other pharmaceutically-acceptable excipients for modifying or maintaining release or absorption or penetration across the blood-brain barrier. Such excipients are those substances usually and customarily employed to formulate dosages for parenteral administration in either unit dosage or multi-dose form or for direct infusion into the cerebrospinal fluid by continuous or periodic infusion.

It is also contemplated that certain formulations containing ADP-expressing vectors are to be administered orally. Such formulations are preferably encapsulated and formulated with suitable carriers in solid dosage forms. Some examples of suitable carriers, excipients, and diluents include lactose, dextrose, sucrose, sorbitol, mannitol, starches, gum acacia, calcium phosphate, alginates, calcium silicate, microcrystalline cellulose, polyvinylpyrrolidone, cellulose, gelatin, syrup, methyl cellulose, methyl- and propylhydroxybenzoates, talc, magnesium, stearate, water, mineral oil, and the like. The formulations can additionally include lubricating agents, wetting agents, emulsifying and suspending agents, preserving agents, sweetening agents or flavoring agents. The compositions may be formulated so as to provide rapid, sustained, or delayed release of the

active ingredients after administration to the patient by employing procedures well known in the art. The formulations can also contain substances that diminish proteolytic degradation and promote absorption such as, for example, surface active agents.

The specific dose is calculated according to the approximate body weight or body surface area of the patient or the volume of body space to be occupied. The dose will also be calculated dependent upon the particular route of administration selected. Further refinement of the calculations necessary to determine the appropriate dosage for treatment is routinely made by those of ordinary skill in the art. Such calculations can be made without undue experimentation by one skilled in the art. Exact dosages are determined in conjunction with standard dose-response studies. It will be understood that the amount of the composition actually administered will be determined by a practitioner, in the light of the relevant circumstances including the condition or conditions to be treated, the choice of composition to be administered, the age, weight, and response of the individual patient, the severity of the patient's symptoms, and the chosen route of administration. Dose administration can be repeated depending upon the pharmacokinetic parameters of the dosage formulation and the route of administration used.

The invention also contemplates passively immunizing patients who have been treated with a viral vector overexpressing ADP. Passive immunization can include administering to the patient antiserum raised against the viral vector, or gamma-globulin or vector-specific purified polyclonal or monoclonal antibodies isolated from the antiserum. Preferably, the patient is passively immunized after a time period sufficient for the viral vector to replicate in and spread through the tumor.

Preferred embodiments of the invention are described in the following examples. Other embodiments within the scope of the claims herein will be apparent to one skilled in the art from consideration of the specification or practice of the invention as disclosed herein. It is intended that the specification, together with the examples, be considered exemplary only, with the scope and spirit of the invention being indicated by the claims which follow the examples.

Example 1

This example illustrates the construction and characterization of the KD1 and KD3 anti-cancer vectors.

To construct KD1, the inventors deleted the entire E3 region of a unique plasmid, leaving behind only a unique PacI site for cloning. The starting plasmid was pCRII, purchased from Invitrogen, containing the Ad5 BamHIA fragment having a deletion of all the

E3 genes; the E3 deletion is identical to that for KD1 and GZ3, the sequences of which are given in SEQ ID NO:1 and SEQ ID NO:4, respectively. The ADP gene from Ad5 was cloned into the PacI site, then built into the E3 region of the genome of the Ad5 E1A mutant named *dI01/07*. This was done by co-transfecting into human embryonic kidney 293 cells the
5 aforementioned BamHIA fragment containing the ADP gene together with the overlapping EcoRIA restriction fragment obtained from *dI01/07*. Complete viral genomes are formed within the cell by overlap recombination between the Ad sequences in the BamHIA fragment in the plasmid and the EcoRIA fragment. KD3 was constructed in the same way except the E3 gene for the 12.5K protein was retained in the starting plasmid. A vector named KD2,
10 which marginally overexpress ADP, was also prepared. Plaques of each recombinant Ad were picked, screened, purified, expanded into CsCl-banded stocks, sequenced, titered, and characterized. GZ1 and GZ3 are Ad vectors that are identical to KD1 and KD3, respectively, except that GZ1 and GZ3 have wild-type E1A sequences as found in AD5 or in the Ad5 mutant *dI309*. GZ1 and GZ3 were constructed as described for KD1 and KD3 except that the
15 EcoRIA fragment of Ad5 was used for GZ1 and GZ3.

KD1 and KD3 were characterized in cell culture by infecting the human A549 lung carcinoma cell line with high titer ($1-8 \times 10^{10}$ plaque forming units [PFU] per ml) virus stocks of one of these recombinant vectors, or with one of the control viruses *dI01/07*, *dI309*, *dI327*, and Ad5 (wt). Fifty PFU per cell were used for each virus. The descriptions of these viruses
20 as well as some other viruses used in these examples are presented in Table 1.

Table 1: Description of mutations in viruses:

Virus	RNA			REGION	
	E1	VA	E3	E4	
<i>d</i> 1101/1107	<i>d</i> 1101: deletion of Ad5 bp 569-634 <i>d</i> 1107: deletion of Ad5 bp 890-928	From <i>d</i> 1309 deletion of Ad5 bp 10594-10595	From <i>d</i> 1309 deletion of Ad5 bp 28597-28602; deletion-substitution Ad5 bp 3005-30750, insert 642 bp DNA of unknown origin	wild type	
KD1	<i>d</i> 1101: deletion of Ad5 bp 569-634 <i>d</i> 1107: deletion of Ad5 bp 890-928	From <i>d</i> 1309 deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 27858-2760, TAA inserted; deletion of Ad5 bp 27982-28134; deletion of Ad5 bp 28395-29397, insert CCTTAATTAAA; deletion of Ad5 bp 29783-30883, insert TTAATTAAAGG	wild type	
KD2	<i>d</i> 1101: deletion of Ad5 bp 569-634 <i>d</i> 1107: deletion of Ad5 bp 890-928	From <i>d</i> 1309 deletion of Ad5 bp 10594-10595	<i>d</i> 1309 background, gp19K mutated deletion of Ad5 bp 28597-28602; deletion-substitution Ad5 bp 3005-30750, insert 642 bp DNA of unknown origin; deletion of Ad5 bp 28788-28789, insert TTAATTAA	wild type	
KD3	<i>d</i> 1101: deletion of Ad5 bp 569-634 <i>d</i> 1107: deletion of Ad5 bp 890-928	From <i>d</i> 1309 deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 28598-29397; deletion of Ad5 bp 29783-30469	wild type	
GZ1	wt	wild type	deletion of Ad5 bp 27858-2760, TAA inserted; deletion of Ad5 bp 27982-28134; deletion of Ad5 bp 28395-29397, insert CCTTAATTAAA; deletion of Ad5 bp 29783-30883, insert TTAATTAAAGG	wild type	

GZ3	wild type	wild type	deletion of Ad5 bp 28598-29397; deletion of Ad5 bp 29783-30469	wild type
<i>d</i> /1101/1107-SPB	<i>d</i> /1101: deletion of Ad5 bp 569-634 <i>d</i> /1107: deletion of Ad5 bp 890-928	From <i>d</i> /309 deletion of Ad5 bp 10594-10595	From <i>d</i> /309 deletion of Ad5 bp 28597-28602; deletion-substitution Ad5 bp 3005-30750, insert 642 bp DNA of unknown origin	E4 promoter deletion-substitution: deletion of Ad5 bp 35623-35775, insert SP-B 500 promoter flanked by BstI 107I sites
KD1-SPB	<i>d</i> /1101: deletion of Ad5 bp 569-634 <i>d</i> /1107: deletion of Ad5 bp 890-928	From <i>d</i> /309 deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 27848-2760, TAA inserted; deletion of Ad5 bp 27982-28134; deletion of Ad5 bp 28395-29397, insert CCTAATTAAA; deletion of Ad5 bp 29783-30883, insert TTAATTAAAGG	E4 promoter deletion-substitution: deletion of Ad5 bp 35623-35775, insert SP-B 500 promoter flanked by BstI 107I sites
KD3-SPB	<i>d</i> /1101: deletion of Ad5 bp 569-634 <i>d</i> /1107: deletion of Ad5 bp 890-928	From <i>d</i> /309 deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 28598-29397; deletion of Ad5 bp 29783-30469	E4 promoter deletion-substitution: deletion of Ad5 bp 35623-35775, insert SP-B 500 promoter flanked by BstI 107I sites

Using a polymerase chain reaction (PCR)-based protocol, an in-frame stop codon was introduced into the gene for the E3-gp19K protein in the E3 region of the Ad5 mutant dl309 (Jones and Shenk, *Cell* 17:683-689, 1979). The mutagenesis was conducted using a *SunI*-*Bst*1107I fragment, nucleotides 28,390 to 29,012 in the Ad5 genome, which was then substituted for the equivalent fragment in *dl*309. *dl*01/07 is the parent for KD1 and KD3. In turn, the Ad5 mutant named *dl*309 is the parent of *dl*01/07, i.e. *dl*309 is identical to *dl*01/07 except that *dl*309 does not have the E1A mutation. Both *dl*01/07 and *dl*309 have deletions of the genes for the E3 RID α , RID β and 14.7K proteins but retain the gene for ADP. The Ad5 mutant *dl*327 has wild-type E1A, it lacks the gene for ADP, and it lacks all other E3 genes except the one for the 12.5K protein.

At 24 and 36 hours post-infection (h p.i.), proteins were extracted from the A549 cells and analyzed for ADP by immunoblot using a rabbit antiserum against ADP (Tollefson et al., *J. Virol.* 66:3633-3642, 1992). The results are shown in Figure 2. Much more ADP was detected at 24 and 36 h p.i. in KD1- and KD3-infected cells than in cells infected with *dl*01/07. Also, much more ADP was synthesized by GZ1 and GZ3 than *dl*309 or the other viruses. Most importantly, KD1, KD3, GZ1, and GZ3 expressed much more ADP at 24 h p.i. than did *dl*01/07 or *dl*309 (Fig. 2). This result is consistent with an observation discussed below that the cells infected with KD1, KD3, GZ1, or GZ3 lyse faster, and that these viruses spread from cell to cell faster than *dl*01/07 or *dl*309. It is noteworthy that KD1, KD3, GZ1, and GZ3 express much more ADP at 24 and 36 h p.i. than the Ad5 mutant *dl*1520 (Fig. 2); *dl*1520 is the original name given to ONYX-015 (Heise et al., *Nature Medicine* 3:639-645, 1997). As expected, no ADP was detected in cells infected with *pm*734.1 (Fig. 2), a mutant that lacks amino acids 1 to 48 in ADP (Tollefson et al., *J. Virol.* 70:2296-2306, 1996). Expression of the E1A proteins by *dl*01/07, KD1, KD2, and KD3 was slightly less than by Ad5, *dl*309, or *dl*327, and as expected from the *dl*01/07 deletion, the proteins were smaller (Fig. 3A). *dl*327 is isogenic with *dl*324 (Thimmappaya et al., 1982 *Cell* 31:543-51, 1983), and it lacks the gene for ADP and all other E3 proteins except the 12.5K protein.

The amount of ADP detected in the KD1 and KD3 infected cells is significantly higher than the amount detected in the *dl*309 infected cells (Fig. 2). If one takes into consideration the fact that the viruses with the E1A mutation replicate somewhat slower, as evidenced in by the delayed appearance of the late proteins (Fig. 3B), it is clear that KD1 and KD3 express much more ADP per viral genome present in the cell than *dl*309. This finding is supported by the fact that when A549 cells are coinfectd with a virus containing the E1A mutation and *dl*327, which lacks ADP but has wild-type E1A, the replication rates of the E1A mutant viruses speed up, as indicated by earlier appearance of late proteins (compare Figs. 3B

and 3D). Thus, *dl327* complements the E1A mutation. In conclusion, these experiments demonstrate that ADP is dramatically overexpressed by KD1, KD3, GZ1, and GZ3. ADP is marginally overexpressed by KD2 (not shown).

5

Example 2

This example illustrates that KD1 and KD3 lyse cells more rapidly and spread from cell-to-cell faster than other adenoviruses.

The ability of KD1 and KD3 to lyse cells was examined by a trypan blue exclusion cell viability assay which was performed essentially as described by Tollefson et al., *J. Virol.* 70:2296-2306, 1996. In brief, A549 cells were mock-infected or infected with 20 PFU/cell of KD1, KD3, *dl01/07*, *dl327* or *dl309*. At various days p.i., the number of viable cells was determined using a hemacytometer (600 to 1000 cells were counted per time point) and the results are shown in Fig. 4.

Only 25% of the KD1-infected cells and 9% of the KD3-infected cells were alive at 5 days p.i. as compared to 44% of cells infected with *dl01/07*, which has the same E1A mutation as KD1 and KD3. The KD1 and KD3 vectors also lysed cells faster than *dl309*, which has a wild-type E1A region. When infected with *dl327* (ADP⁺, E1A⁺), 94% of the cells were alive after 5 days. When cell lysis was estimated by release of lactate dehydrogenase, KD1 and KD3 once again lysed cells faster than *dl01/07* and *dl309*, and *dl327* caused little cell lysis (data not shown). Thus, ADP is required for efficient cell lysis, and over-expression of ADP increases the rate of cell lysis.

As another means to measure cell lysis and to examine virus replication in cancer cells, separate groups of A549 cells were infected with 20 PFU/cell of KD1, KD3, *dl01/07*, or *dl309* and the amount of intracellular and extracellular virus was determined by plaque assay on A549 cells. At 2 days p.i., the total amount of virus formed in each group was similar, $2-4 \times 10^8$ PFU/ml, indicating that replication of all the viruses is similar. However, when the ratio of extracellular to intracellular virus was calculated, the value for KD1 and KD3 was 2-3 logs higher than for Ad5, *dl309*, or *dl01/07* (data not shown). Thus, virus is released much more rapidly from cells infected with KD1 and KD3, which overexpress ADP, than with viruses expressing wild-type amounts of ADP.

The ability of KD1 and KD3 to spread from cell-to-cell was measured in a "cell spreading" assay. In this assay monolayers of A549 cells in a 48 well culture dish were mock-infected or infected with 10^{-3} , 10^{-2} , 10^{-1} , 10^0 , or 10 PFU/cell of *dl327*, *dl309*, Ad5, *dl01/07*, KD1 or KD3. At low PFU/cell, the viruses must go through two or three rounds of replication in order to infect every cell in the monolayer. At 1.0 and 10 PFU/cell, the

monolayer should be destroyed by the virus that initially infected the cells. To assess the amount of spread in the monolayers by 7 days p.i., crystal violet, which stains live cells but not dead cells, was added to the monolayers. The results are shown in Fig. 5.

Remarkably, at 7 days p.i., the monolayer was virtually eliminated by KD1 and KD3 at 10^{-3} PFU/cell, whereas 1.0 PFU/cell was required with *dl*01/07, *dl*309 and Ad5. This result attests to the potency of ADP in mediating cell lysis and virus spread in A549 cells. KD1 and KD3 are also more effective than *dl*01/07 in killing other types of human cancer cell lines (most purchased from the American Type Culture Collection [ATCC]) as determined in this cell spreading assay. KD1 and/or KD3 killed HeLa (cervical carcinoma), DU145 (prostate), and pC3 (prostate) cells at 10^{-2} PFU/cell, ME-180 (cervix) and Hep3B (liver) at 10^{-1} PFU/cell, and U118 (glioblastoma) and U373 (glioblastoma) at 10 PFU/cell. From 10- to 100-fold more *dl*01/07 was required to kill these cells (data not shown). These results indicate that KD1 and KD3 may be effective against many types of cancer.

An important aspect of the finding that ADP overexpressing vectors lyse cells at very low multiplicities of infection is that the multiplicity of infection in human tumors is likely to be low at sites distal to the site of vector injection or distal to blood vessels that carry the vector to the tumor. Thus, ADP overexpressing vectors have an advantage over vectors that express less ADP or no ADP at all.

Example 3

This example illustrates that KD1 and KD3 replicate poorly in non-growing non-cancerous cells. The replication phenotype of KD1 and KD3 was evaluated using "normal" HEL-299 human fibroblast cells, either growing in 10% serum or rendered quiescent using 0.1% serum. All Ads should replicate well in growing cells, but viruses with the *dl*01/07 E1A mutation should do poorly in quiescent cells because E1A is required to drive them out of G_0 . *dl*309, which has wild-type E1A, should replicate well in both growing and growth-arrested cells.

Cells were infected with 100 PFU/cell of KD1, KD3, *dl*01/07, or *dl*309. At different days p.i., virus was extracted and titered. In 10% serum, KD1, KD3, and *dl*01/07 replicated well, reaching titers of 10^6 - 10^7 PFU/ml, only slightly less than *dl*309 (Fig. 6). However, in quiescent cells, replication of KD1, KD3, and *dl*01/07 was 1.5-2 logs lower than in growing cells, ranging from 10^4 to 2×10^5 PFU/ml. The titer of *dl*309 reached 10^7 PFU/ml, nearly the level achieved in growing cells. At 10 days p.i., quiescent HEL-299 cell monolayers infected with 100 PFU/cell of KD1, KD3, or *dl*01/07 were intact, whereas those infected with *dl*309 or *dl*327, which have wild-type E1A, showed strong typical Ad cytopathic effect indicative of

cell death (data not shown). Thus, replication of KD1 and KD3 is severely restricted to growing cell lines.

The restriction associated with the *dl01/07* E1A mutation was also tested in primary human cells (purchased from Clonetics) growing as monolayers. Bronchial epithelial cells (Fig. 7) and small airway epithelial cells were not killed by 10 PFU/cell of KD1, KD3, or *dl01/07* at 5 days p.i., whereas they were killed by 10 PFU/cell of *dl309* or *dl327* (data not shown). Lung endothelial cells also were not killed after 10 days by KD1, KD3, or *dl01/07* at 10 PFU/cell, but they were killed by 1 PFU/cell of *dl309*. These monolayers were subconfluent when initially infected, then grew to confluency. The exciting result here is that although these primary cells were growing, they did not support replication in this time frame and were not killed by KD1 or KD3. Thus, it is believed these vectors will be restricted to cancerous cells, and will have little to no effect on cells such as basal cells that are normally dividing in the body. In addition, it is unlikely that KD1 and KD3 will affect dividing leukocytes because such cells are poorly infected by Ad.

In summary, the above experiments demonstrate that KD1 and KD3 lyse cancer cells, spread from cell-to-cell rapidly, and replicate poorly in quiescent and non-cancerous cells. These properties should make them useful in anti-cancer therapy.

Example 4

This example illustrates that KD1 and KD3 inhibit the growth of human tumors in an animal model.

We could not evaluate mouse or rat tumors in normal mice or rats because they are totally non-permissive. Human cancer cell lines growing in nude mice have been used by Onyx Pharmaceuticals (Richmond, CA) to evaluate the efficacy of ONYX-015, an Ad vector lacking expression of the E1B 55 kDa protein (Heise et al., *Nature Med.* 3:639-645, 1997). We have found that A549 cells, which were used in many of our cell culture studies, form excellent rapidly growing solid tumors when injected subcutaneously into nude mice. The average tumor reaches ca. 500 μ l in four weeks, and is encapsulated, vascularized, and attached to the mouse skin (usually) or muscle.

Nude mice were inoculated into each hind flank with 2×10^7 A549 cells. After 1 week tumors had formed, ranging in size from about 20 μ l to 50 μ l. Individual tumors were injected three days later, and at subsequent weeks for 4 weeks (total of 5 injections), with 50 μ l of buffer or 50 μ l of buffer containing 5×10^7 PFU of *dl309*, *dl01/07*, KD1, KD3, or *pm734.1*, with a total virus dose per tumor of 3×10^8 PFU. The mutant *pm734.1* lacks ADP activity due to two nonsense mutations in the gene for ADP, but all other Ad proteins are

expected to be synthesized at wild-type levels (Tollefson et al., *J. Virol.* 70:2296-2306, 1996). The efficacy of each virus (or buffer) was tested on six tumors. At weekly intervals, the length (L) and width (W) of tumors were measured using a Mitutoyo digital caliper. Tumor volumes were calculated by multiplying $L \times W \times W/2$. This value was divided by the tumor volume at the time of the initial virus injection, the fold-increase in tumor growth was calculated, and the average for the six tumors was graphed.

As shown in Fig. 8A, tumors that received buffer continued to grow, increasing about 14-fold by 5 weeks. In contrast, tumors injected with *dl309*, which expresses normal amounts of ADP and lacks the E3 RID and 14.7K and proteins, only grew about 2.5-fold by 5 weeks. With *pm734.1*, which lacks ADP, the tumors grew as well as those that received buffer. Thus, *dl309* markedly decreases the rate of tumor growth, and ADP is required for this decrease. Tumors inoculated with *dl01/07* grew about 8-fold over 5 weeks. Since *dl01/07* is identical to *dl309* except for the E1A mutation, this result indicates that the E1A mutation significantly reduces the ability of Ad to prevent growth of the tumors. This effect is probably due to a reduction in virus replication in the tumors resulting in lower ADP expression, but it could also reflect other properties of E1A in the tumor cells, e.g. the inability of the mutant E1A proteins to induce apoptosis. Most importantly, tumors inoculated with KD1 or KD3 only grew about 2.5-fold. Thus, the overexpression of ADP by KD1 and KD3 allows KD1 and KD3 to reduce tumor growth to a rate markedly slower than *dl01/07* (their parental control virus), and even to a rate similar to that of *dl309*.

The finding that KD1 and KD3 are as effective as wild-type Ad (i.e. *dl309*) in reducing the rate of A549 tumor growth is highly significant in the context of cancer treatment, inasmuch as KD1 and KD3 are restricted to cancer cells whereas wild-type Ad does not have such a restriction.

The tumors in Fig. 8A received five injections of vectors, but only one dose of vector, in this case 5×10^8 of each of KD3 or GZ3, is sufficient to significantly reduce the rate of A549 tumor growth (Fig. 8B).

We have also found that KD1 and KD3 reduce the rate of growth in nude mice of a human liver cancer cell line, Hep3B cells. These cells form rapidly growing tumors that are highly vascularized. Nude mice were inoculated into each hind flank with 1×10^7 of Hep3B cells. After tumors reached about 100 μ l, they were injected twice per week for 3 weeks with 50 μ l of buffer or 5×10^7 PFU of KD1, KD3, or *dl309*. There were typically 8-10 tumors per test virus. The tumor sizes were measured and the fold increase in size at 0 to 3.5 following the initial virus injection was graphed as described above for the A549 tumors. Tumors that received buffer alone grew 9-fold over 3 weeks and were projected to grow about 12-fold

over 3.5 weeks (after 3 weeks the mice had to be sacrificed because the tumors were becoming too large) (Fig. 9). Tumors that received KD1 or KD3 grew about 4-fold, establishing that KD1 and KD3 reduce the growth of Hep3B tumors in nude mice. Tumors that were injected with *dl309* grew 2-fold (Fig. 9). The finding that KD1 and KD3 were somewhat less effective than *dl309* is probably due to the fact that they do not grow as well as *dl309* in Hep3B cells, as indicated by a cell spread assay in culture (data not shown). In any case, the important points are that KD1 and KD3 are effective against the Hep3B tumors, and that they contain the E1A mutation that limits their replication to cancer cells.

These results point to the potency of ADP as an anti-tumor agent when expressed in an Ad vector. It is highly probable that KD1 and KD3 will provide significant clinical benefit when used to infect tumors growing in humans.

Example 5

This example illustrates the use of replication-defective Ad vectors in combination with KD1 or KD3.

It is well established that replication-competent (RC) viruses complement replication-defective (RD) mutants. That is, when the same cell is infected, the competent virus will supply the protein(s) that cannot be made from the mutant genome, and both viruses will grow. To test the ability of KD1 and KD3 to complement RD viruses, two RD vectors expressing β -galactosidase were constructed. The first, named Ad- β -gal, has a cDNA encoding β -gal under the control of the Rous Sarcoma Virus promoter substituted for the deleted E1 region. Ad- β -gal also has the E3 region deleted, including the gene for ADP. The second, named Ad- β -gal/FasL is identical to Ad- β -gal, except that it also expresses murine FasL from the human cytomegalovirus promoter/enhancer. These vectors were constructed by overlap recombination in human 293 cells that constitutively express the Ad E1A and E1B genes and complement replication of the E1-minus vectors.

These RD vectors should infect and express β -gal in A549 cells, but should not replicate because the E1A proteins are lacking. However, the vectors should replicate when cells are co-infected with RC Ads. To prove this, A549 cells were infected with 10 PFU/cell of Ad- β -gal alone, or with 10 PFU/cell of Ad- β -gal plus 10 PFU/cell of KD1, KD3, *dl01/07*, *dl309*, or *dl327*. At 2 days p.i., virus was extracted and Ad- β -gal titers determined by β -gal expression in A549 cells. The yields are shown in Table 2 below.

Table 2

Virus	Yield (blue plaques per ml)
Ad- β -gal	1×10^2
Ad- β -gal + KD1	2×10^5
Ad- β -gal + KD3	3×10^5
Ad- β -gal + <i>dl01/07</i>	4×10^4
Ad- β -gal + <i>dl309</i>	3×10^5
Ad- β -gal + <i>dl327</i>	3.0×10^5

The data in Table 2 indicate that the complementing viruses increased the yield of Ad- β -gal by about 10^3 .

5 A key feature of KD1 and KD3 is that they spread from cell-to-cell faster than other Ads. Accordingly, they should complement the spread of Ad- β -gal. To test this, an infectious center assay was conducted. A549 cells were infected with Ad- β -gal plus KD1, KD3, or *dl01/07*. After 2 h, cells were collected, diluted, and seeded onto monolayers of fresh A549 cells. After 4 days, the cells were stained with X-gal and the results are shown in
10 Fig. 10.

With Ad- β -gal alone, only the originally infected cell (before seeding) should be stained, and the vector should not spread to other cells on the seeded monolayer. This was indeed the case. In monolayers seeded with A549 cells infected with Ad- β -gal alone (dish shown in the top left of Fig. 10A) contained a number of individual blue cells (not visible in
15 the print); examples are shown in the enlarged view Fig. 10B. However, when the monolayers were seeded with A549 cells coinfecting with Ad- β -gal and KD1 or KD3, there were numerous "comets" of blue cells (Fig. 10A). Each comet represents Ad- β -gal which has spread from one initially-infected cell. Most of the cells within a comet were stained with X-gal (Fig. 10C). Comets were also observed with *dl01/07*, but not to the extent of KD1 and
20 KD3 (Fig. 10A). With *dl327* (ADP⁻), there was little spread from the originally infected cell (data not shown). In summary, KD1 and KD3 not only complement the replication of Ad- β -gal, they also enhance its rapid spread.

It is expected that KD1 and KD3 will also complement and enhance the spread of RD vectors expressing anti-cancer therapeutic gene products, and this expectation can be readily

verified using the Ad- β -gal/FasL in replication and infectious center assays as described above.

KD1 and KD3 not only complement the replication of RD vectors in cell culture, they also do so in Hep3B tumors growing in the hind flanks of nude mice. The RD vector used was AdLuc, an Ad that lacks the E1 and E3 regions, and has inserted into the E1 region an expression cassette where the firefly luciferase gene is expressed from the Rous Sarcoma Virus promoter (Harrod et al., *Human Gene Therapy* 9:1885-1898, 1998). The Hep3B tumors were injected with 1×10^7 PFU of AdLuc plus buffer, or 1×10^7 PFU of AdLuc plus 5×10^7 PFU of KD1, KD3, *dI01/07*, or *dI309*. After 2 weeks, mice were sacrificed and tumors excised. Proteins were extracted from the tumors and luciferase activity determined using a luminometer. The luciferase counts per tumor were 6,800 for AdLuc plus buffer, 113,500 for KD1, and 146,900 for KD3 (Fig. 11). Thus, KD3 and KD1 respectively caused a 22-fold and 17-fold increase in luciferase activity. This increase could be due to elevated synthesis of luciferase in cells that were initially coinfecting the AdLuc and KD1 or KD3, and it could also be due to spread of AdLuc from cell to cell in the tumor as mediated by KD1 or KD3.

In summary, infecting a tumor with a replication-competent ADP-overexpressing vector according to the invention together with a RD vector expressing an anti-cancer gene product should greatly increase the amount of anti-cancer protein synthesized in the tumor thereby increasing the ability of the replication-defective vector to promote destruction of the tumor.

Example 6

This example illustrates the construction and characterization of a recombinant Ad vector according to the invention which is replication-restricted to cancerous type II alveolar cells.

As demonstrated above, the *dI01/07* mutation in KD1 and KD3 limits growth of these vectors to cancer cells. To further restrict their replication phenotype, the E4 promoter in each virus was deleted and replaced by the surfactant protein B (SPB) promoter to produce vectors named KD1-SPB (SEQ ID NO:14), KD3-SPB (SEQ ID NO:15), and *dI01/07*-SPB (SEQ ID NO:16). The SPB promoter is only active in cells containing the TTF1 transcription factor, which has thus far been found primarily in type II alveolar cells of the human lung (Lazzaro et al., *Development* 113:1093-1104, 1991). Thus, KD1-SPB, KD3-SPB, and *dI01/07*-SPB should be severely restricted to cancerous type II alveolar cells of the human lung. Many lung cancers are of this type.

The KD1-SPB and KD3-SPB vectors were prepared as follows. The E4 promoter is located at the right end of the Ad genome (Fig. 1). Using a pCRII-based plasmid (Invitrogen) containing the Ad5 DNA sequences from the BamHI site (59 map units) to the right hand end of the genome, and using a PCR-based protocol, nearly all the transcription factor binding sites were deleted from the E4 promoter Ad5 base pairs 35,623 to 35,775 and replaced with a 500 base pair fragment containing the SPB promoter (Yan et al., *J. Biol. Chem.* 270:24852-24857, 1995). The final plasmids contain the E4-SPB substitution in the E4 region and the *dl01/07*, KD1, or KD3 versions of the E3 region, respectively, for the viruses *dl01/07*-SPF, KD1-SPB, and KD3-SPB. These plasmids were co-transfected into 293 cells with a fragment containing the left portion of the genome of *dl01/07*, and plaques were allowed to develop. Plaques were screened for the expected features, purified, then expanded into a stock.

The A549-TTF1 cell line was developed in order to test the prediction that replication of *dl01/07*-SPB, KD1-SPB, and KD3-SPB would be restricted to cancerous cells expressing the TTF1 transcription factor. These cells were co-transfected with two plasmids, one in which TTF1 is expressed from the CMV promoter, and the other coding for resistance to neomycin. Resistant clones were isolated and shown to express TTF1 activity as determined by transient transfection with a plasmid expressing chloramphenicol acetyltransferase from the TTF1-requiring surfactant protein C promoter.

KD1-SPB and KD1 were subjected to a standard plaque development assay on A549-TTF1 cells and parental A549 cells. The results are shown in Fig. 12. With KD1-SPB on A549 cells, plaques were not visible after 8 days, only about 4% of the final number of plaques were seen after 10 days, and about 50% of final plaques were seen after 12 days. With KD1-SPB on A549-TTF1 cells, plaques were visible after 6 days, and about 60% of plaques were seen after 10 days. Thus, as expected, KD1-SPB grew significantly faster on the cells containing TTF1. KD1 formed plaques more quickly than KD1-SPB on both A549 and A549-TTF1 cells, indicating that the E4 promoter-SPB substitution is not as effective as the wild-type E4 promoter in inducing Ad replication. However, this difference between KD1-SPB and KD1 on A549-TTF1 cells is tolerable, with KD1-SPB delayed only about 1 day. Curiously, the final titer obtained for all virus stocks by day 16 was similar, indicating that A549 cells may contain a very small amount of endogenous TTF1 activity. It is predicted that KD3-SPB and *dl01/07*-SPB will behave similarly to KD1-SPB when grown in A549-TTF1 cells and A549 cells.

The restriction of KD1-SPB to cells containing TTF1 was further examined in a cell spread assay using H441 cells, a TTF1-expressing human pulmonary adenocarcinoma cell line (Yan et al., *supra*), and Hep3B cells, a liver cancer cell line not expected to express

TTF1. Culture dish wells containing H441 or Hep3B cells were infected with KD1-SPB or KD1 at multiplicities ranging from 10 to 10^{-4} PFU/cell. The H441 and Hep3B cells were stained with crystal violet at 5 days and 8 days p.i., respectively. KD1-SPB and KD1 grew and spread equally well on H441 cells, causing destruction of the monolayer at 10^{-1} PFU per cell (Fig. 13). (Some of the H441 monolayer has peeled off in the well with KD1-SPB at 10^{-2} PFU per cell, and in the wells with KD1 and KD1-SPB at 10^{-4} PFU per cell; this occasionally occurs in cell spread assays, and it does not reflect virus infection). With Hep3B cells, KD1 grew and spread very much better than KD1-SPB, with 10^{-2} PFU per cell of KD1 causing more destruction of the monolayer as 1.0 PFU per cell of KD1-SPB (Fig. 13).

In summary, this example demonstrates that a replication-competent Ad, which replicates well on cells expressing the appropriate transcription factor, can be constructed with a tissue-specific promoter substituted in place of the E4 promoter. This methodology should be applicable to many other tissue specific and cell type specific promoters. One possibility would be a liver-specific promoter. Another possibility would be to use the E2F promoter, or another promoter with E2F sites, inasmuch as that promoter would be active only in cells such as cancer cells that have free E2F. A third possibility would be to use a regulatable promoter, e.g. the synthetic tetracycline response promoter (Massie et al., *J. Virol.* 72:2289-2296, 1998), where the activity of the promoter is controlled by the level of tetracycline or a tetracyclin analog in the patient.

Example 7

This example illustrates the construction and characterization of vectors which overexpress ADP and are not replication restricted.

As demonstrated above, the *dI01/07* E1A mutation in KD1 and KD3 is attenuating, inhibiting growth in non-dividing and even in dividing primary human epithelial and endothelial cells. Ads with this mutation are able to replicate well in dividing cancer cells. However, replication of such E1A mutants is not as efficient as, e.g. *dI309* which has a wild-type E1A gene. For instance, the rate of replication of *dI01/07*, as determined by the rate at which plaques develop, is reduced such that *dI01/07* plaques appear one day later than those of *dI309* (data not shown). This delay is due in part to a delay in expression of Ad late genes (see Fig. 3). The idea that the *dI01/07* mutation retards the rate of replication in A549 cells is further supported by the data in Fig. 8A, where *dI01/07* did not prevent tumor growth nearly as well as *dI309*. Despite this negative effect of the *dI01/07* E1A mutation, there are theoretical and practical aspects of having this mutation in the KD1 and KD3 vectors, as has been discussed. Nevertheless, one can easily imagine scenarios (e.g. patients with terminal

cancer) where the ability of an Ad vector to destroy the tumor supercedes the requirement that the vector be totally restricted to tumor cells. In such cases, it would be advantageous to have vectors similar to KD1 and KD3, but with the wild-type E1A gene. The rates at which such vectors express their genes, lyse cells, and spread from cell to cell should be higher than those of KD1 and KD3. Such vectors might cause some damage to non-cancerous cells and tissue, but this is also true for other modes of anti-cancer treatment such as surgery, chemotherapy, and radiation therapy.

In light of these considerations, vectors named GZ1 and GZ3 have been constructed that are identical to KD1 and KD3, respectively, except they have a wild-type E1A region.

These vectors were constructed by overlap recombination in A549 cells. The left hand fragment contained the wild-type E1A region of Ad5, and the right end fragment contained the E3 modifications of KD1 or KD3. Plaques were picked, analyzed for the expected genotype, plaque-purified, and expanded into CsCl-banded stocks. The titers of these stocks on A549 cells were 2.9×10^{10} PFU/ml for GZ1 and 1.6×10^{11} PFU/ml for GZ3. Thus, these vectors can be grown into high titer stocks comparable to wild-type Ad. The GZ1 and GZ3 plaques are larger and appear much sooner than the plaques for *dl309*. Large rapidly-appearing plaques reflect the ability of Ad to lyse cells and spread from cell-to-cell (Tollefson et al., *J. Virol.* 70:2296-2306, 1996; Tollefson et al., *Virology* 220:152-162, 1996), and this property, as discussed, is due to the function of ADP.

The rate of plaque appearance can be quantitated in a plaque development assay (Tollefson et al., *supra*). Here, a typical plaque assay is performed, and the plaques observed on subsequent days of the assay are calculated as a percentage of the number of plaques observed at the end of the plaque assay. As shown in Fig. 14, after 4 days of plaque assay on A549 cells, GZ1 and GZ3 had 48% and 34%, respectively, of the final number of plaques, whereas *dl309* had only 1%. It is very unusual in Ad plaque assays in A549 cells for plaques to appear after only 4 days. These large plaques reflect the overexpression of ADP. These GZ1 and GZ3 plaques appear sooner than those of KD1 and KD3 (data not shown), no doubt because GZ1 and GZ3 replicate faster because they have a wild-type E1A region.

GZ1 and GZ3 lyse cells and spread from cell to cell much more effectively than *dl309*. At 6 days p.i. of A549 cells, approximately as much monolayer destruction was observed with GZ1 and GZ3 at 10^{-3} PFU per cell as was observed with *dl309* at 10^{-1} PFU per cell (Fig. 15, top panel). This result further underscores the conclusion that overexpression of ADP promotes cell lysis and virus spread.

In theory, GZ1 and GZ3 should be able to replicate not only in tumor cells but also in normal cells. Although they can replicate in normal cells, it is quite possible that GZ1 and

GZ3 may be useful as anti-cancer vectors. First, GZ1 and GZ3 could be injected directly into the tumor. Many tumors are self-contained (encapsulated) except for the blood supply. The physical barriers of the tumor could minimize dissemination of the virus to other tissues. Second, Ads are in general quite benign. Most infections of Ad5 are in infants and result in mild or asymptomatic disease, and are held in check by strong humoral and cellular immunity. Anti-Ad immunity appears to be life-long. GZ1 and GZ3 could be used only in patients who have an intact immune system, and perhaps also with pre-existing anti-Ad immunity. Further, patients could be passively immunized against Ad, using gamma-globulin or even specific purified anti-Ad neutralizing antibodies. Third, considering that Ad5 is a respiratory virus which most efficiently infects lung epithelial cells displaying the specific Ad5 receptor (named CAR) as well as specific integrins (e.g. $\alpha_v \beta_5$), replication-competent vectors derived from Ad5 may not spread efficiently in many non-cancer tissues of the body. In addition, it is believed that versions of GZ1 and GZ3 can be constructed that have the E4 promoter substituted with a tumor-specific, tissue-specific, cell-specific, or synthetic promoter. Such vectors would have the positive features associated with wild-type E1A and ADP, and yet be replication-restricted to tumor tissue and/or to particular cell types.

Example 8

This example illustrates that the combination of KD1, KD3, GZ1, or GZ3 with radiation is more effective in destroying A549 cells, growing in culture or growing as tumors in nude mice, than the vectors alone or radiation alone.

This was shown in a cell spread assay. A549 cells growing in three 48 well culture dishes were mock-infected or infected with different viruses at multiplicities of infection ranging from 10 to 10^{-4} PFU per cell as indicated in Fig. 15. One dish was not radiated. A second dish received 600 centigrays (cGy) of radiation at 24 h p.i., and a third dish received 2000 cGy of radiation at the same time. All dishes were stained with crystal violet at 6 days p.i. With the cells that were not radiated (top panel in Fig. 15), KD1 and KD3 caused monolayer destruction at lower multiplicities of infection than their parental control, *dl101/07*. This was also true for GZ1 and GZ3 as compared to their parental control *dl309*. (The paucity of cells in the cells infected with GZ1 or GZ3 at 10^{-4} PFU per cell is an experimental artifact, and is not caused by infection by GZ1 or GZ3). These KD1, KD3, GZ1 and GZ3 results are consistent with earlier results showing that overexpression of ADP leads to increased cell lysis and virus spread.

With the dish that was infected then radiated with 600 cGy there was markedly increased cell killing and virus spread as compared to the non-radiated cells (compare the

bottom panel of Fig. 15 with the top panel). For example, with KD1, KD3, GZ1, and GZ3 there was about the same amount of cell destruction in the radiated wells at 10^{-4} PFU per cell as in the non-radiated wells at 10^{-2} PFU per cell. Similar results were seen with the dish that received 2000 cGy of radiation (data not shown), and also with dishes that received 600 or 2000 cGy of radiation 24 h prior to infection (data not shown).

The amount of cell destruction was quantitated by extracting the crystal violet from the cells with 33% acetic acid, then measuring the absorbance at 490 nm (data not shown). The absorbance with non-radiated mock-infected cells was set at 100% cell viability. With mock-infected cells that received 600 cGy there was a 15% loss in viability (i.e. 15% less crystal violet was extracted). With KD1 at 10^{-3} PFU per cell, the non-radiated cells were 80% viable whereas the cells receiving 600 cGy of radiation were only about 30% viable. Similar differences in viability between radiated and non-radiated cells were seen with KD3, GZ1, and GZ3. These results argue that the combination of radiation plus vector has a synergistic effect on cell lysis and vector spread, rather than an additive effect. If the effect were only additive, then with the KD1 samples at 10^{-3} PFU per cell, the cell viability should have been 65% (15% reduction in viability due to radiation alone, 20% reduction due to KD1 alone). In fact, the cell viability was 30% rather than 65%.

As mentioned, approximately as much cell lysis and virus spread were observed with 600 cGy as with 2000 cGy. To determine the optimal dose of radiation to synergize with the vectors, an experiment similar to the one described above was conducted with mock-, *dI01/07*-, KD1-, KD3-, *dI309*-, GZ1-, or GZ3-infected A549 cells. The 48 well plates received 0, 150, 300, or 600 cGy of radiation at 24 h p.i. Cells were stained with crystal violet. The results with cells receiving 0 versus 600 cGy of radiation were similar to those in Fig. 15. The crystal violet was extracted from the cells infected with 10^{-3} PFU per cell of the difference viruses. The absorbance of crystal violet was determined, and the percent cell viability was graphed, using the absorbance of the non-radiated mock-infected cells as 100% cell viability. As illustrated in Fig. 16, an approximately linear decrease in cell viability in all wells was obtained with increasing radiation dose, although the slope of the line was more negative with KD1, KD3, GZ1, or GZ3 than with mock, *dI01/07*, or *dI309*. With KD1, KD3, GZ1, and GZ3, there was much more cell lysis and vector spread with their parental control viruses, and there was synergy between the vectors and radiation. For example, with mock-infected cells, 600 cGy reduced cell viability by about 30% (70% of cells were viable). KD1 without radiation reduced cell viability by about 23%. The combination of 600 cGy radiation plus KD1 reduced cell viability to about 85%, more than 53% of which is the sum of radiation

alone and KD1 alone. When considering the data in Figs. 15 and 16 together, a dose of about 600 cGy is optimal in this type of cell culture experiment.

The combination of KD3 or GZ3 with radiation was also examined in the A549 tumor-nude mouse model (see Example 4). A549 cells were injected into the hind flanks of nude mice, and tumors were allowed to form. When tumors reached approximately 50- μ l, they were injected with buffer or with 5×10^8 PFU of KD3 or GZ3. Eight to ten tumors were injected per test condition. At 1 day p.i., half the mice received 600 cGy of whole body radiation. Tumor size was measured over time, and was plotted as a fold-increase in tumor size versus days p.i. (as described in Example 4). As shown in Fig. 17, the non-radiated buffer-injected tumors grew faster than those injected with KD3 or GZ3. Tumors that received the combination of KD3 and radiation did not grow, and those that received the combination of GZ3 and radiation shrank in size after 14 days. These results indicate that the combination of KD3 plus radiation or GZ3 plus radiation is more effective than either vector alone or radiation alone in reducing the rate of A549 tumor growth in nude mice. It is likely that radiation would increase the effectiveness in treating tumors of KD1 and GZ1, or indeed any other replication-competent or replication-defective Ad vector.

The mechanism by which radiation causes the ADP overexpressing vectors to lyse cells and spread from cell-to-cell more effectively is not understood. Radiation is expected to induce cellular DNA repair mechanisms, and that may allow for more efficient synthesis of Ad DNA. Radiation may enhance the function of ADP. ADP probably functions by interacting with one or more cellular proteins, and radiation may affect this protein(s) such that ADP functions more efficiently.

It is believed that KD1, KD3, GZ1, or GZ3, or any other replication-competent Ad vector, when used in combination with radiation, will be more effective than vector alone or radiation alone in providing clinical benefit to patients with cancer. The vectors should allow more tumor destruction with a given amount of radiation. Stated another way, radiation should cause more tumor destruction with a given amount of vector. These vectors should also allow the radiation oncologist to use less radiation to achieve the same amount of tumor destruction. Less radiation would reduce the side effects of the radiation.

It is also believed that a cocktail of vectors when used in combination with radiation will be more effective than the cocktail alone or radiation alone. The cocktail could consist of ADP producing vectors plus one or more replication defective vectors expressing an anticancer therapeutic protein (see Example 5).

Example 9

This example illustrates a structure-function analysis of adenovirus death protein.

ADP is an 11.6 kDa N-linked O-linked integral membrane glycoprotein that localizes to the inner nuclear membrane (NM) (Scaria et al., *Virology* 191:743-753). As illustrated in Fig. 18, the Ad2-encoded ADP (SEQ ID NO:6) consists of 101 amino acids; aa 1-40 (SEQ ID NO:17) are luminal, aa 41-59 (SEQ ID NO:18) constitute the transmembrane signal-anchor (SA) domain, aa 63-70 (SEQ ID NO:19) constitute a basic proline (BP) domain within the nucleoplasmic (NP) domain, which constitutes aa 61-101 (SEQ ID NO:20). To determine which domains in ADP are required to promote cell death, a number of deletion mutants of *rec700* were prepared which lacked various portions of the ADP gene and examined for the ability of ADP to localize to the NM and promote death. The *rec700* virus is an Ad5-Ad-Ad5 recombinant which has been described elsewhere (Wold et al., *Virology* 148:168-180, 1986).

The structure of ADP in *rec700* and in each deletion mutant is schematically illustrated in Fig. 18. The ADP gene in each deletion mutant has been sequenced using PCR methods to insure that the mutations are correct. The structure and activity of ADP in the deletion mutants was tested by infecting A549 cells followed by immunoblot analysis of the ADP mutant proteins as well as the ability to lyse cells. All deletion mutants expressed a stable ADP protein except *pm734.1* ($\Delta 1-48$, i.e. aa 1-48 are deleted). The *pm734.7* (N_{14}) ADP, which has Asn_{14} mutated to Ser, is O-glycosylated but not N-glycosylated because Asn_{14} is the only N-glycosylation site (data not shown). The *dl735* ($\Delta 4-11$) ADP is N-glycosylated but not O-glycosylated because the sites for O-glycosylation are deleted (data not shown). The *pm734.4* (M_{56}) ADP, which has Met_{56} in the SA domain mutated to Ser, contains exclusively N-linked high-mannose oligosaccharides (data not shown); this occurs because the Met_{56} mutation precludes exit of ADP from the endoplasmic reticulum (ER). The *dl738* ADP, which lacks aa 46-60 in the signal-anchor domain, forms insoluble aggregates in the cytoplasm; therefore, aa 41-59 do in fact include the signal-anchor domain. The *pm734* ($\Delta 1-40$) ADP, which initiates at Met_{41} at the N-terminus of the SA domain, comigrated with the lower group of bands generated by proteolytic processing (data not shown). This indicates that the proteolytic cleavage sites occur near Met_{41} . Consistent with this, the proteolytic products were not seen with *dl737* ($\Delta 29-45$) (data not shown). Also, the size of the products decreased in all mutants with deletions within aa 41-101 (*dl715.1*, *dl715*, *dl714*, *dl716*) (data not shown).

The ability of these mutants to promote cell death was monitored by trypan blue exclusion, plaque development, and lactate dehydrogenase release assays (Tollefson et al., *J. Virol.* 70:2296-2306, 1996). The trypan blue results in Fig. 15A indicate that the death-

promoting function of ADP was abolished by deletion of aa 1-40 (*pm734*), aa 11-26 (*dl736.1*), aa 18-22 (*dl735.1*), or aa 4-11 (*dl735*). Mutation of the N-glycosylation site at Asn₁₄ (*pm734.7*) reduced the death-promoting activity to about 50% of *rec700* (WT). *dl737* (Δ 29-45) was efficient as *rec700* in promoting cell death; this indicates that the proteolytic processing products must not be required to promote cell death because they are not formed with *dl737*. The SA domain is essential for death because *dl738* (Δ 46-60) and *pm734.4* (M56) were completely defective (Fig. 19). *dl715.1* was nearly completely defective, indicating that the BP domain is extremely important. Surprisingly, aa 71-94 (*dl714*), 76-89 (*dl715*), and 79-101 (*dl716*) could be deleted without affecting the death-promoting activity of ADP (Fig. 19). On the other hand, deletion of aa 81-88 (*dl717*) nearly completely abolished the activity of ADP (Fig. 19); this is probably the result of aberrant sorting of ADP (see below). Similar results were obtained when the ability of these ADP mutants to promote cell death was examined with standard plaque development, LDH-release and MTT assays.

The effects of these mutations on the intracellular localization of ADP are extremely interesting. When examined by immunofluorescence (IF) at 33 h p.i. (data not shown), ADP from *rec700* (WT) localized crisply to the NM; localization to the Golgi was also apparent. With *dl714* (Δ 71-94) and *dl715* (Δ 76-89), ADP localized to all membranes, i.e. the ER, Golgi, plasma membrane, and NM. This was even more apparent at 45 h p.i. (data not shown). Thus, aa 71-94 appear to include a signal that directs ADP specifically to the NM. ADP is very likely sorted from the *trans*-Golgi network (TGN) to the NM, so this putative signal in ADP probably functions in this sorting pathway. ADP from *dl717* (Δ 81-88) is intriguing: it localized to the NM and Golgi, but in many cells "dots" and circular structures were observed. Again, this was more apparent at 45 h p.i. when these structures were the prominent feature. *dl717*-infected cells have not begun to die at 45 h p.i., so these structures are not cellular remnants. The intriguing possibility is that these structures are membrane vesicles that have pinched off from the TGN but are defective in targeting to and/or fusing with the NM.

With *dl738* (Δ 46-60 in the SA domain), ADP aggregated in the cytoplasm. This again indicates that aa 46-60 include the SA sequence. With *pm734.4* (M56), ADP localized primarily to the NM. As discussed above, the *pm734.4* ADP has exclusively high-mannose N-linked oligosaccharides, indicating that it never leaves the ER. Perhaps the putative NM-localization signal in the C-terminal region of the *pm734.4* ADP targets ADP to the NM by lateral diffusion from the ER (which is continuous with the outer and inner NM).

With *dl737* (Δ 29-45), ADP localized to the NM. ADP from *pm734* (Δ 1-40), *pm734.7* (N14) (N-linked glycosylation cannot occur), and *dl735* (Δ 4-11; the O-glycosylation sites are deleted) localized much more prominently to the Golgi than the NM. ADP from *dl735.1*

(Δ 18-22) and *dl736.1* (Δ 11-26) also localized much more strongly to the Golgi than the NM. Thus, residues 1-26 and/or glycosylation appear to be required for efficient transport of ADP from the Golgi/TGN to the NM.

In summary, aa 41-59 include the SA domain, Met₅₆ in the SA domain is required for exit from the ER, aa 1-26 are required for efficient exit from the Golgi, and aa 76-94 are required to target ADP specifically to the NM. With respect to promoting cell death, the essential regions are aa 1-26, the SA domain (ADP does not enter membranes), Met₅₆ in the SA domain, and the BP domain (aa 63-70). It is not clear whether the defective death-promoting phenotype of *pm734* (Δ 1-40), *dl735* (Δ 4-11), *dl735.1* (Δ 18-22), *dl736.1* (Δ 11-26), and *pm734.7* (N14) is due to lack of sequences (or oligosaccharides) that promote death or to much slower exit of ADP from the Golgi to the NM. *dl714* (Δ 71-94) and *dl715* (Δ 76-89) express a wild-type phenotype for promoting death even though they are defective in localizing specifically to the NM; this is probably because sufficient ADP still enters the NM to promote death. Even though the deletion in *dl717* (Δ 81-88) lies within the deletions in *dl715* (Δ 76-89) and *dl714* (Δ 71-94), the *dl717* ADP is only about 15% as efficient as *rec700* (WT), *dl715* and *dl714* in promoting death. This may be because the *dl717* ADP tends to remain in vesicles rather than localizing to the NM. Altogether, these data indicate that ADP must localize to the NM in order to promote cell death.

In view of the above, it will be seen that the several advantages of the invention are achieved and other advantageous results attained.

As various changes could be made in the above methods and compositions without departing from the scope of the invention, it is intended that all matter contained in the above description and shown in the accompanying drawings shall be interpreted as illustrative and not in a limiting sense.

All references cited in this specification, including patents and patent applications, are hereby incorporated by reference. The discussion of references herein is intended merely to summarize the assertions made by their authors and no admission is made that any reference constitutes prior art. Applicants reserve the right to challenge the accuracy and pertinency of the cited references.

What is Claimed Is:

1. A recombinant vector which is replication-competent in a neoplastic cell and which overexpresses an adenovirus death protein.
2. The recombinant vector of claim 1 wherein the adenovirus death protein comprises amino acids 1-26, 41-59, and 63-70 of SEQ ID NO:5, SEQ ID NO:6, SEQ ID NO:7, or SEQ ID NO:8 or a conservatively substituted variant thereof or wherein the adenovirus death protein comprises SEQ ID NO:5, SEQ ID NO:6, SEQ ID NO:7, or SEQ ID NO:8.
3. The recombinant vector of claim 2 which comprises a recombinant virus.
4. The recombinant vector of claim 3, wherein the recombinant virus is an adenovirus lacking expression of at least one E3 protein selected from the group consisting of: gp19K; RID α ; RID β and 14.7K.
5. The recombinant vector of claim 4 which comprises SEQ ID NO:3 or SEQ ID NO:4.
6. The recombinant vector of claim 3 which is replication-restricted to neoplastic cells.
7. The recombinant vector of claim 6 which comprises SEQ ID NO:1 or SEQ ID NO:2.
8. The recombinant vector of claim 3, wherein the recombinant adenovirus comprises a tissue specific promoter or an inducible promoter substituted for the E4 promoter.
9. The recombinant vector of claim 6 which comprises SEQ ID NO:14, SEQ ID NO:15 or SEQ ID NO:16.
10. A method for promoting death of a neoplastic cell comprising contacting the neoplastic cell with at least one vector which is replication competent in the neoplastic cell and which overexpresses an adenovirus death protein.

11. The method of claim 10 wherein the adenovirus death protein comprises amino acids 1-26, 41-59, and 63-70 of SEQ ID NO:5, SEQ ID NO:6, SEQ ID NO:7, or SEQ ID NO:8 or a conservatively substituted variant thereof or wherein the adenovirus death protein comprises SEQ ID NO:5, SEQ ID NO:6, SEQ ID NO:7, or SEQ ID NO:8.

12. The method of claim 11, wherein the vector comprises a recombinant adenovirus lacking expression of at least one E3 protein selected from the group consisting of: gp19K; RID α ; RID β and 14.7K.

13. The method of claim 12, wherein the neoplastic cell comprises a tumor in a patient and the contacting step comprises administering the recombinant adenovirus to the tumor.

14. The method of claim 13, further comprising the step of passively immunizing the patient against the recombinant adenovirus.

15. The method of claim 14, wherein the recombinant adenovirus comprises SEQ ID NO:3 or SEQ ID NO:4.

16. The method of claim 12, wherein the vector is replication-restricted to neoplastic cells.

17. The method of claim 16, wherein the vector is a recombinant adenovirus comprising SEQ ID NO:1 or SEQ ID NO:2.

18. The method of claim 12, wherein the recombinant adenovirus comprises a tissue specific promoter or an inducible promoter substituted for the E4 promoter.

19. The method of claim 18, wherein the recombinant adenovirus which comprises SEQ ID NO:14, SEQ ID NO:15 or SEQ ID NO:16.

20. The method of claim 13; further comprising treating the tumor with radiation.

21. The method of claim 20 comprising administering more than one recombinant adenovirus to the tumor and treating the tumor with radiation.

22. The method of claim 13, further comprising treating the tumor with chemotherapy.

23. The method of claim 22 comprising administering more than one recombinant adenovirus to the tumor and treating the tumor with chemotherapy.

24. The method of claim 13, further comprising administering to the tumor one or more replication-defective adenovirus which expresses an anti-cancer gene product, wherein the recombinant adenovirus complements spread of the replication-defective adenovirus in the tumor.

25. A composition comprising:

a first recombinant virus which is replication competent in a neoplastic cell and overexpresses an adenovirus death protein; and

5 a second recombinant virus which is replication defective and which expresses an anti-cancer gene product,
wherein the first recombinant virus complements replication of the second recombinant virus.

26. The composition of claim 25 wherein the first recombinant virus comprises a recombinant adenovirus lacking expression of at least one E3 protein selected from the group consisting of: gp19K; RID α ; RID β and 14.7K.

27. The composition of claim 26 wherein the recombinant adenovirus comprises a nucleotide sequence selected from the group consisting of: SEQ ID NO:1; SEQ ID NO:2; SEQ ID NO:14; SEQ ID NO:15; SEQ ID NO:16; SEQ ID NO:3; or SEQ ID NO:4.

REPLICATION-COMPETENT ANTI-CANCER VECTORS

Abstract of the Disclosure

- Novel vectors which are replication competent in neoplastic cells and which overexpress an adenovirus death protein are disclosed. Some of the disclosed vectors are
- 5 replication-restricted to neoplastic cells or to neoplastic alveolar type II cells. Compositions and methods for promoting the death of neoplastic cells using these replication-competent vectors are also disclosed.

0994778-071299



ADP Is Expressed Earlier in Infection By KD1, KD3, GZ1, and GZ3

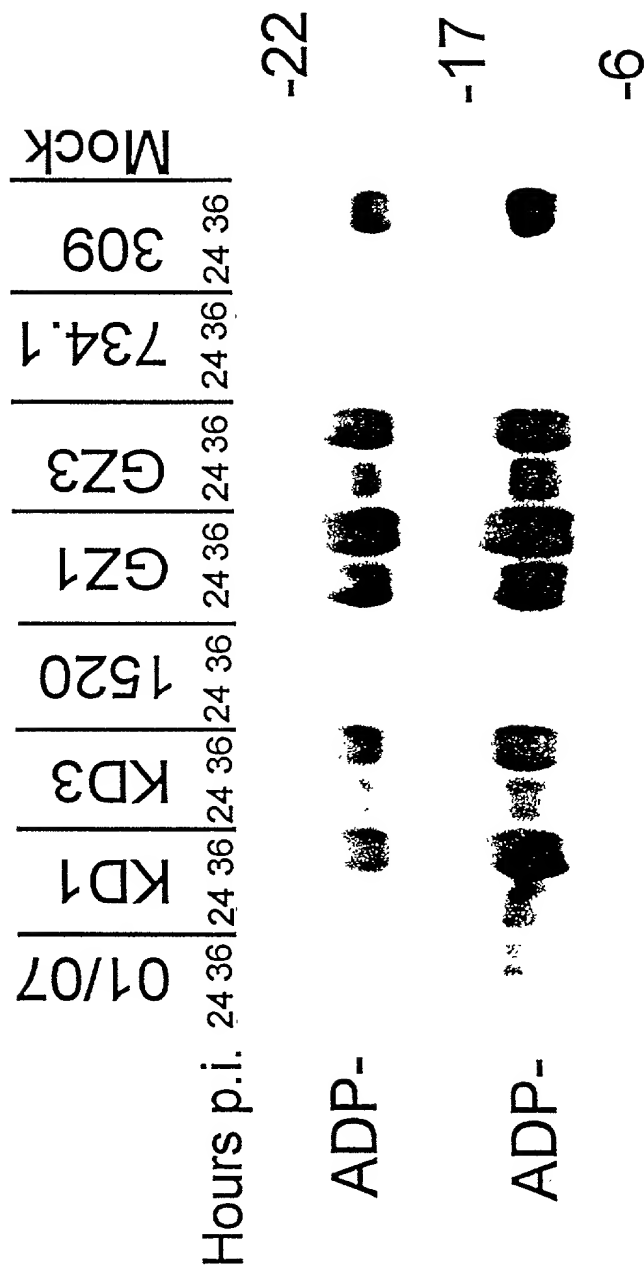


FIGURE 2

The E1A 01/07 Mutation Retards Late Gene Expression

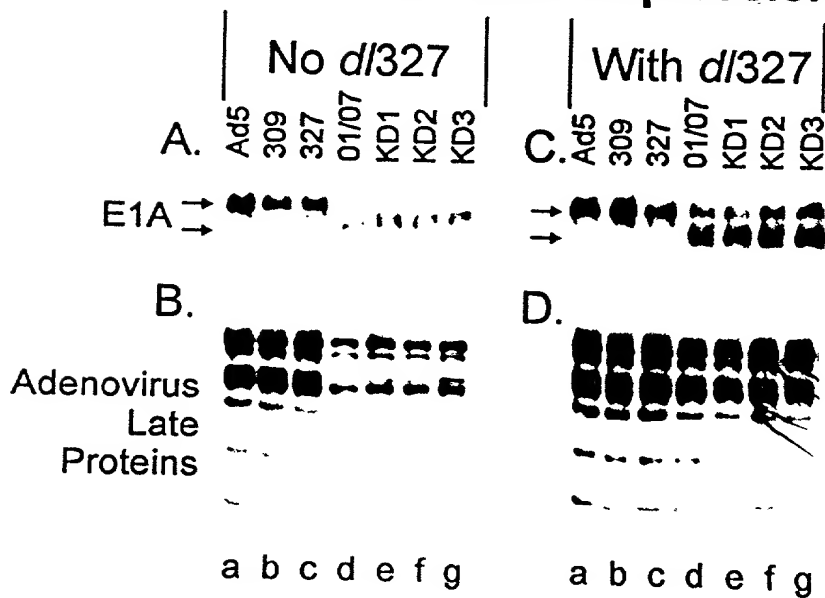


FIGURE 3

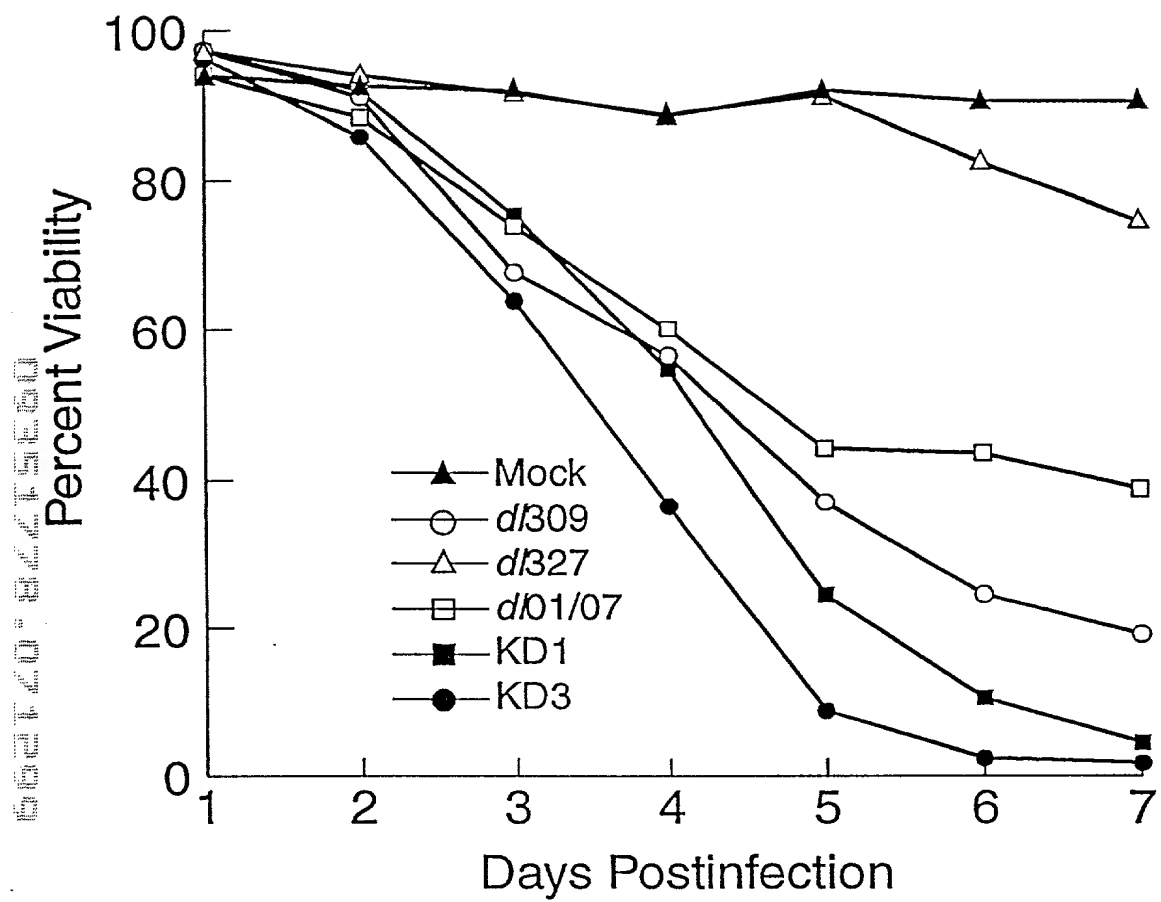


FIGURE 4

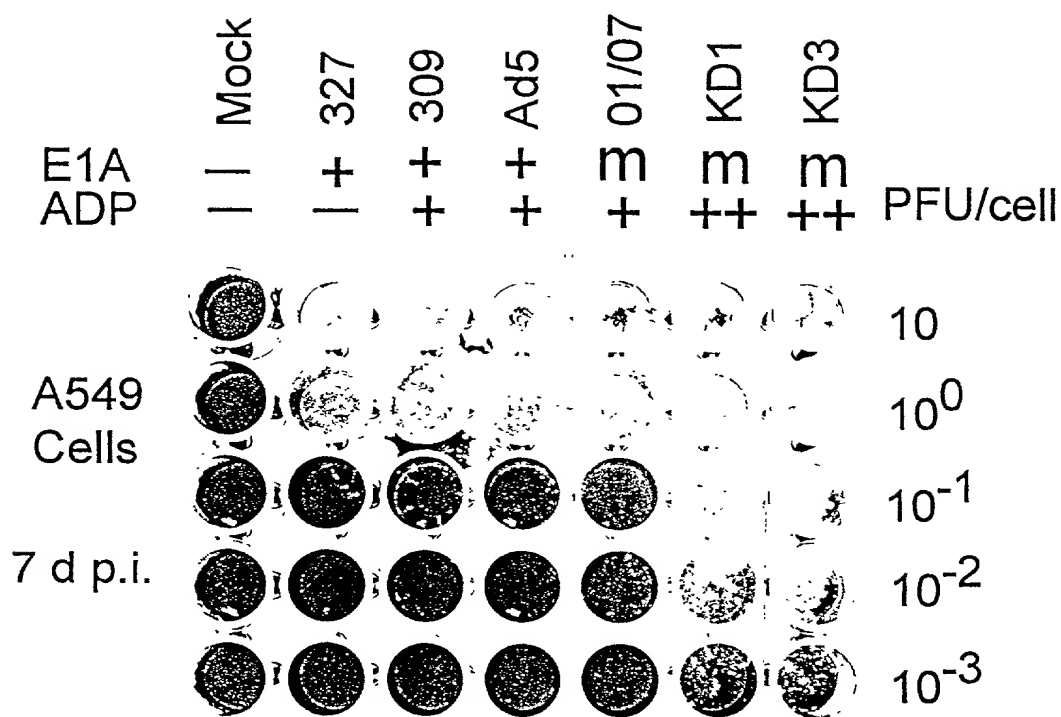


FIGURE 5

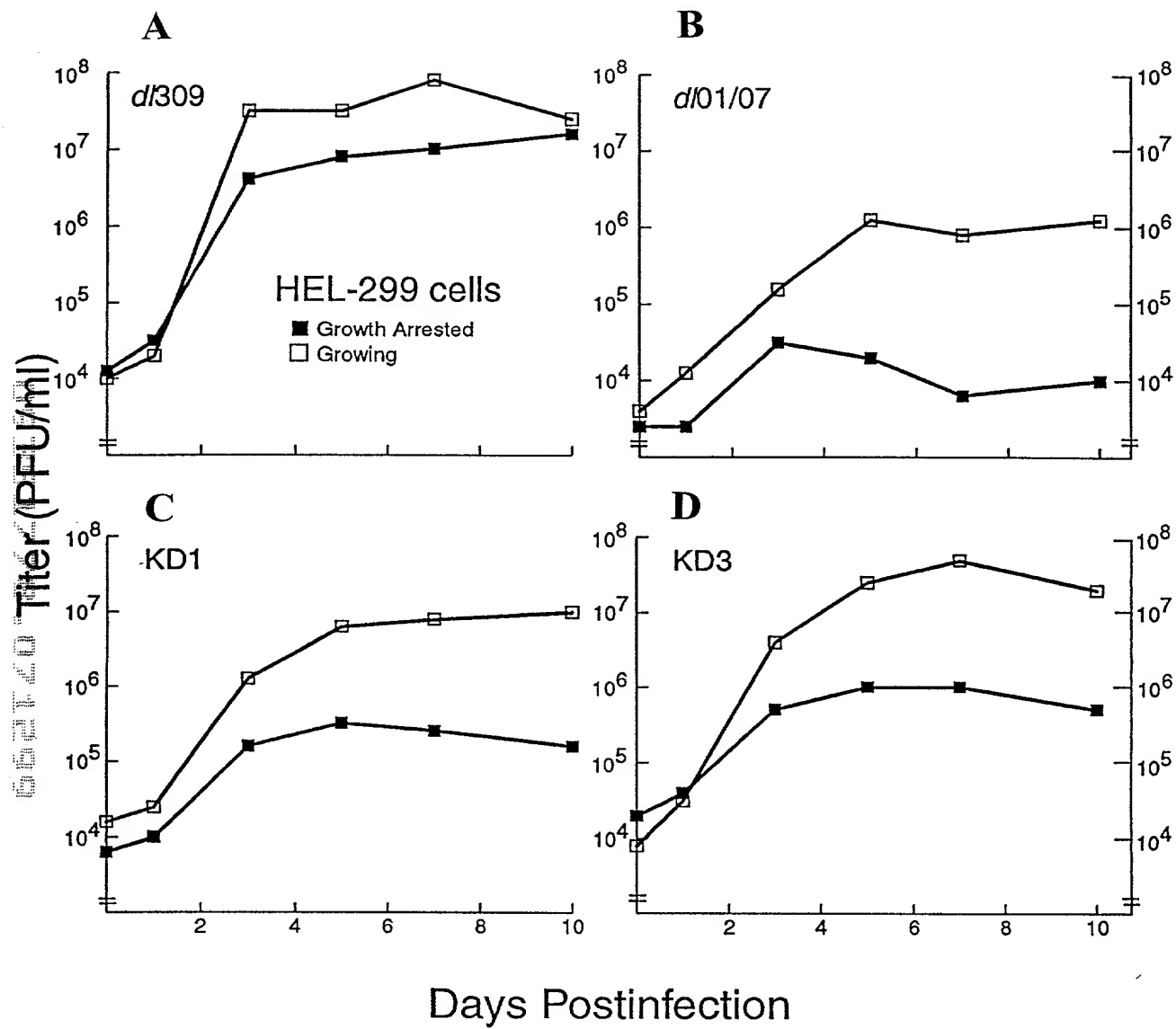
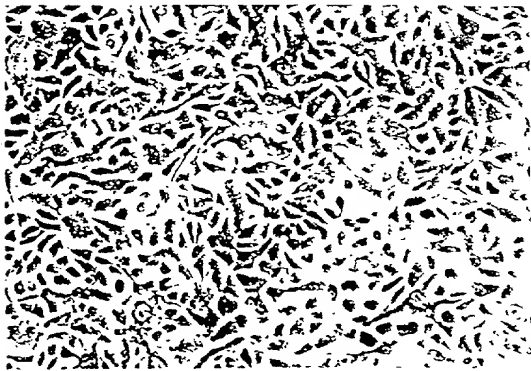
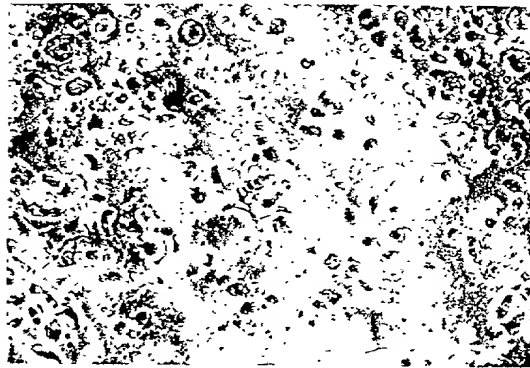


FIGURE 6

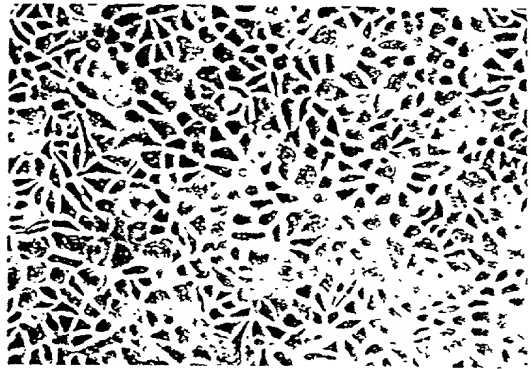
Mock



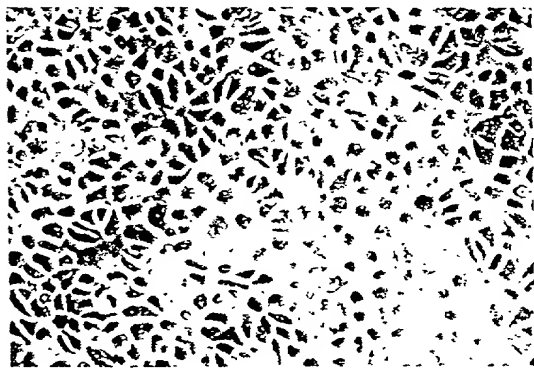
309 (E1A⁺, ADP⁺)



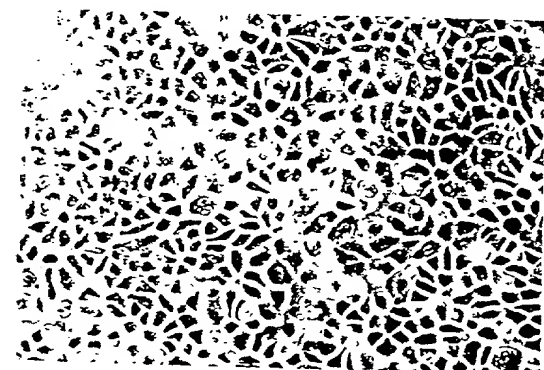
01/07 (E1A^m, ADP⁺)



KD1 (E1A^m, ADP⁺⁺)



KD3 (E1A^m, ADP⁺⁺)



327 (E1A⁺, ADP⁻)

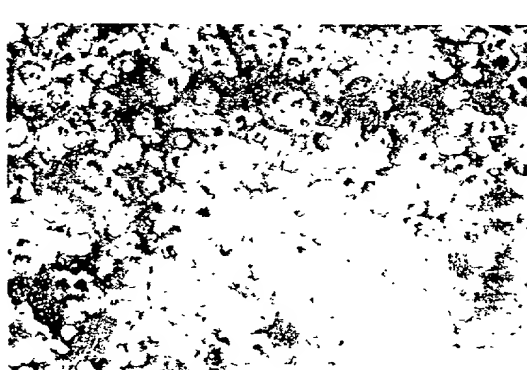


FIGURE 7

662720-8445860

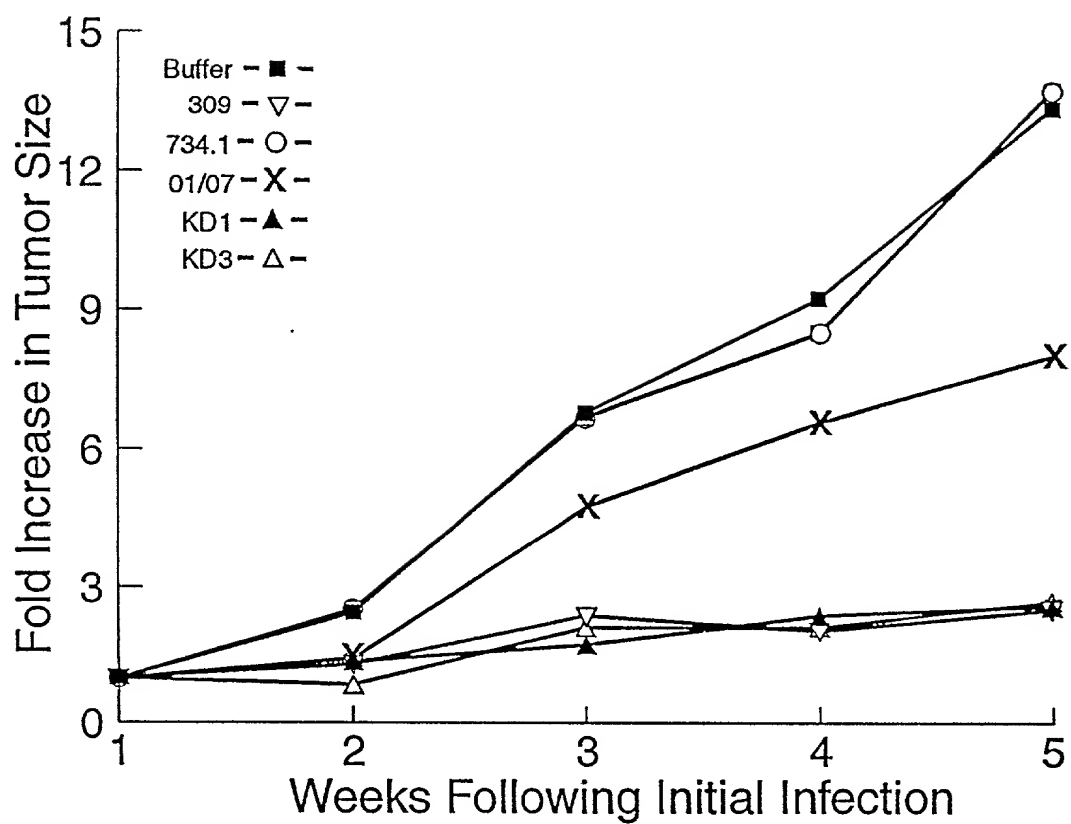


FIGURE 8A

One Injection of KD3 or GZ3 Inhibits
Growth of A549 tumors
(5×10^8 PFU injected on day 0)

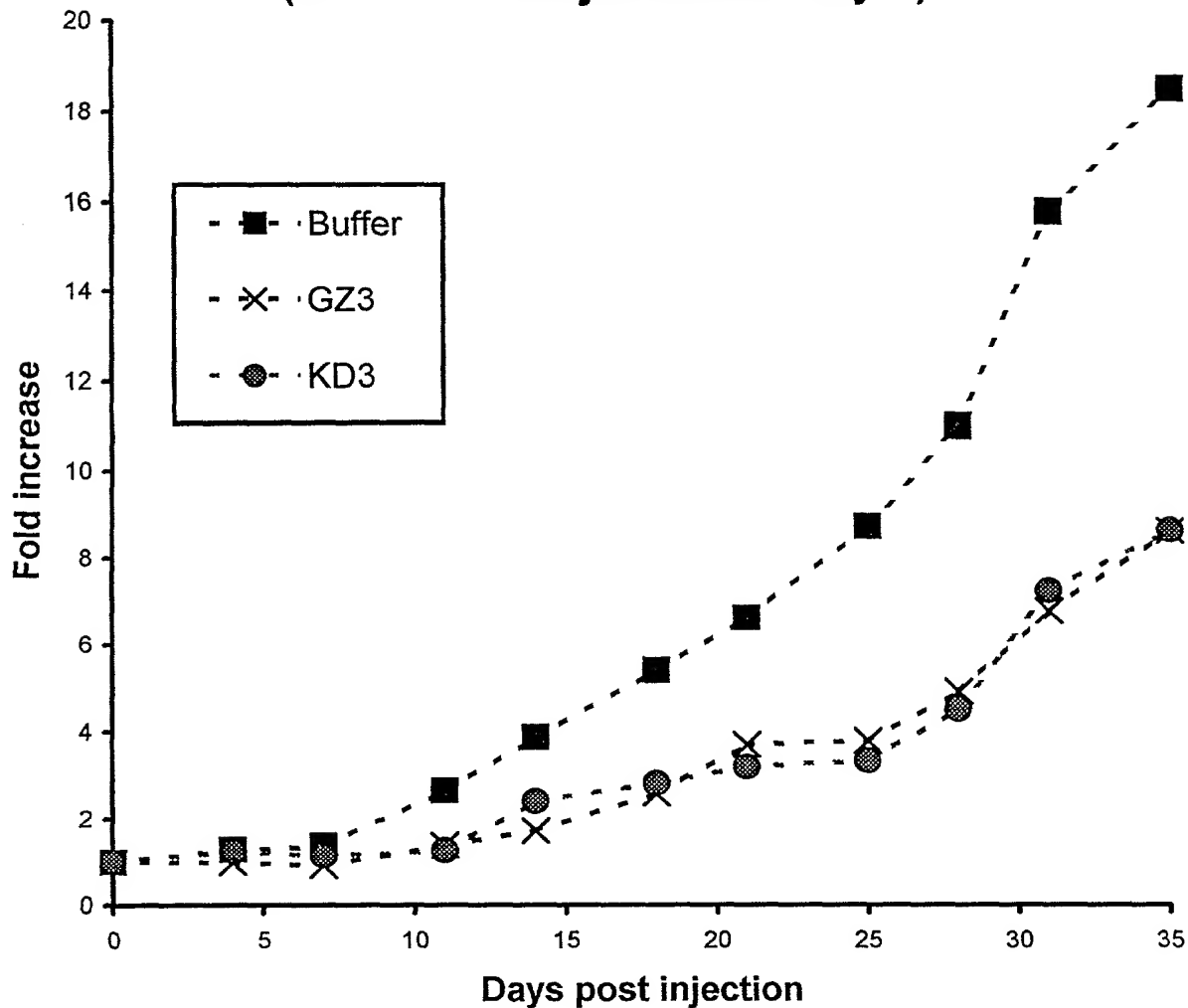


FIGURE 8B

662720 8447360

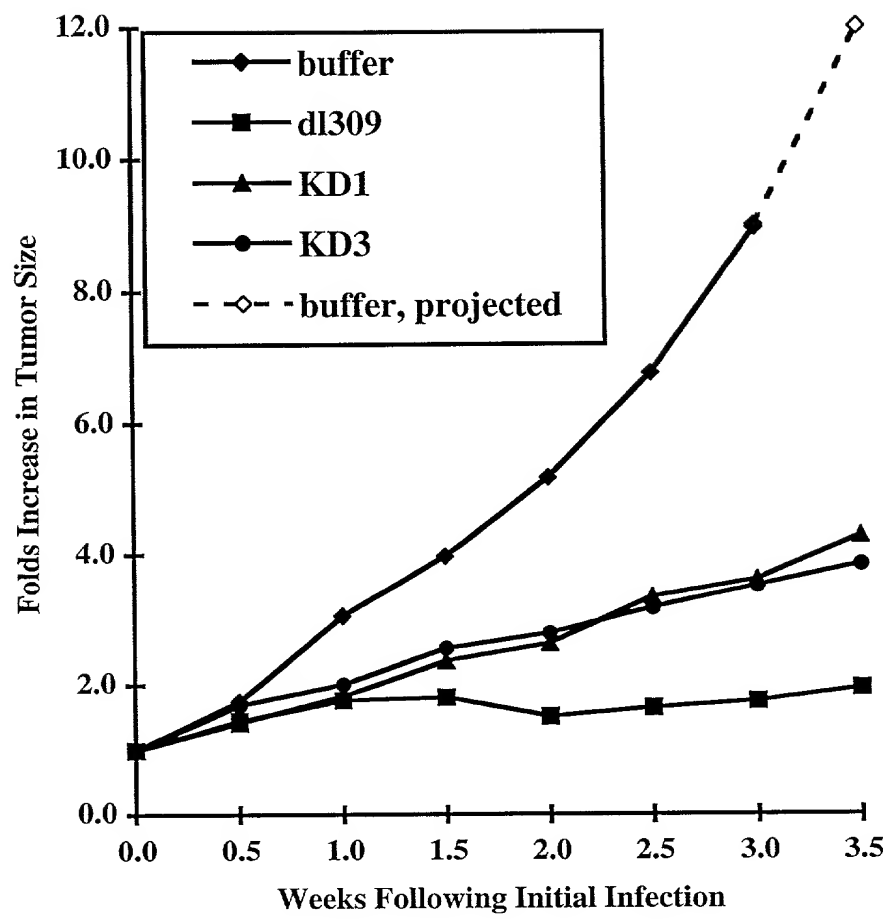


FIGURE 9

Ad- β -gal Alone Ad- β -gal+*dl01/07*

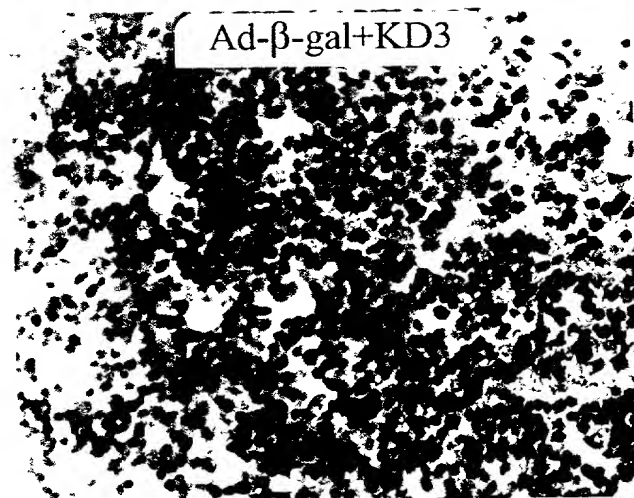
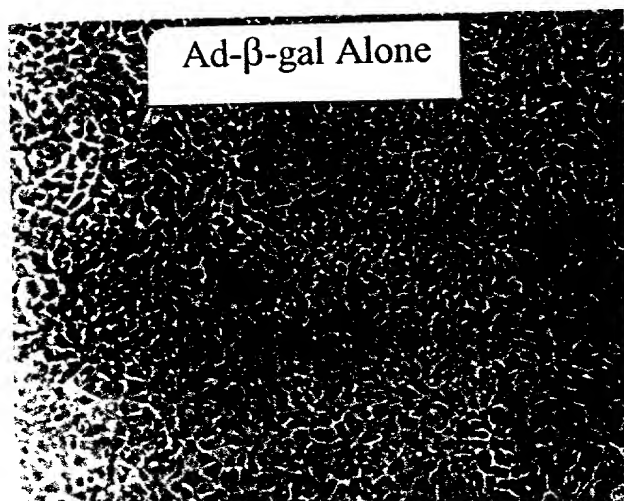
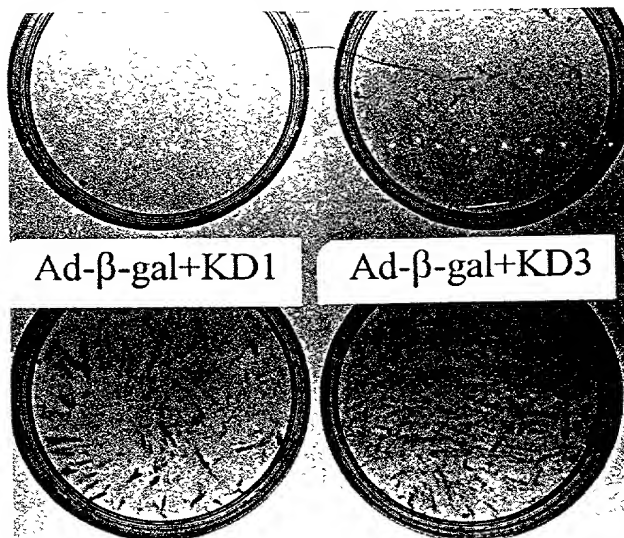


FIGURE 10

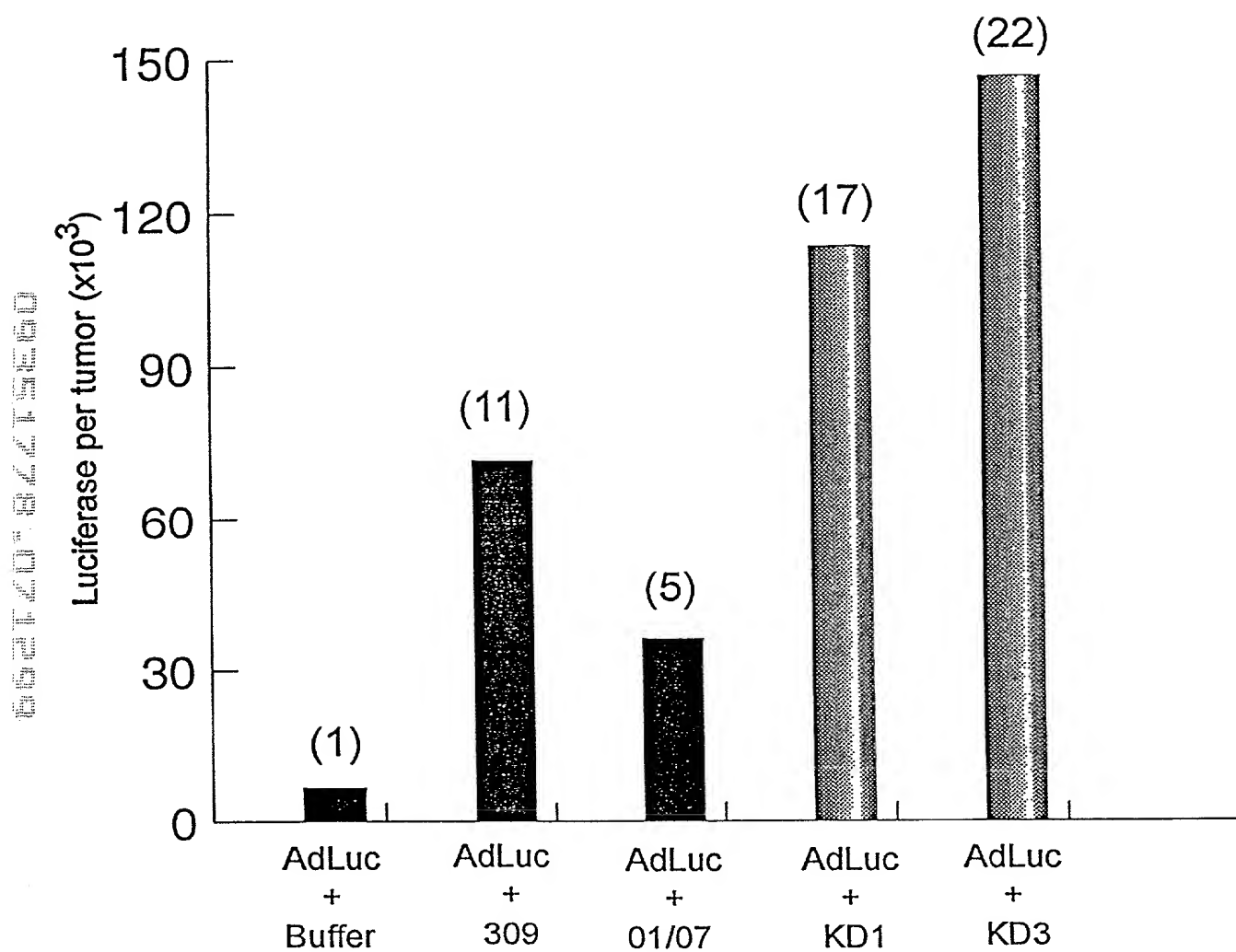


FIGURE 11

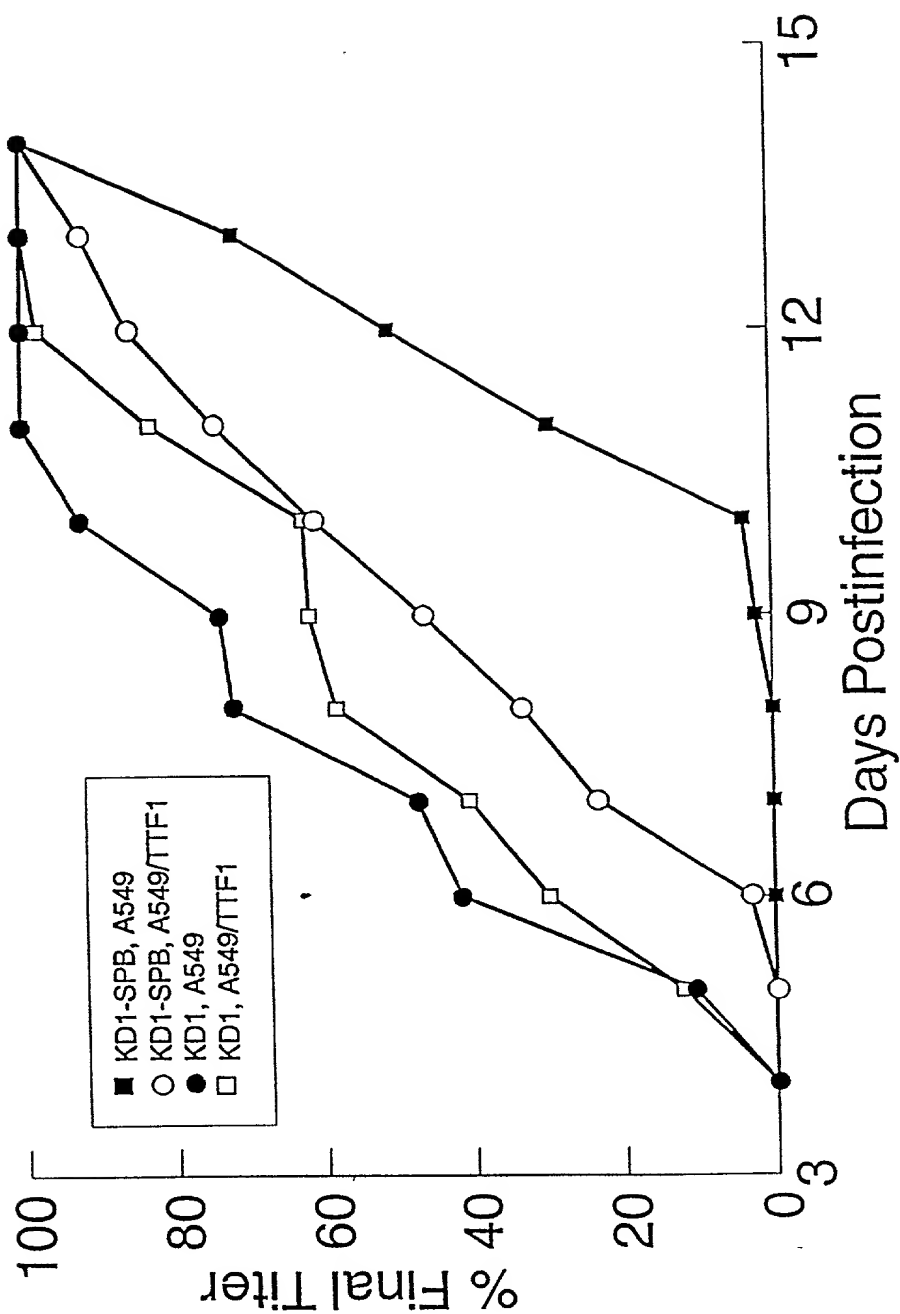


FIGURE 12

KD1-SPB With the SPB Promoter in Place of the E4 Promoter Grows on H44a Lung Cancer Cells with the TTF1 Transcription Factor

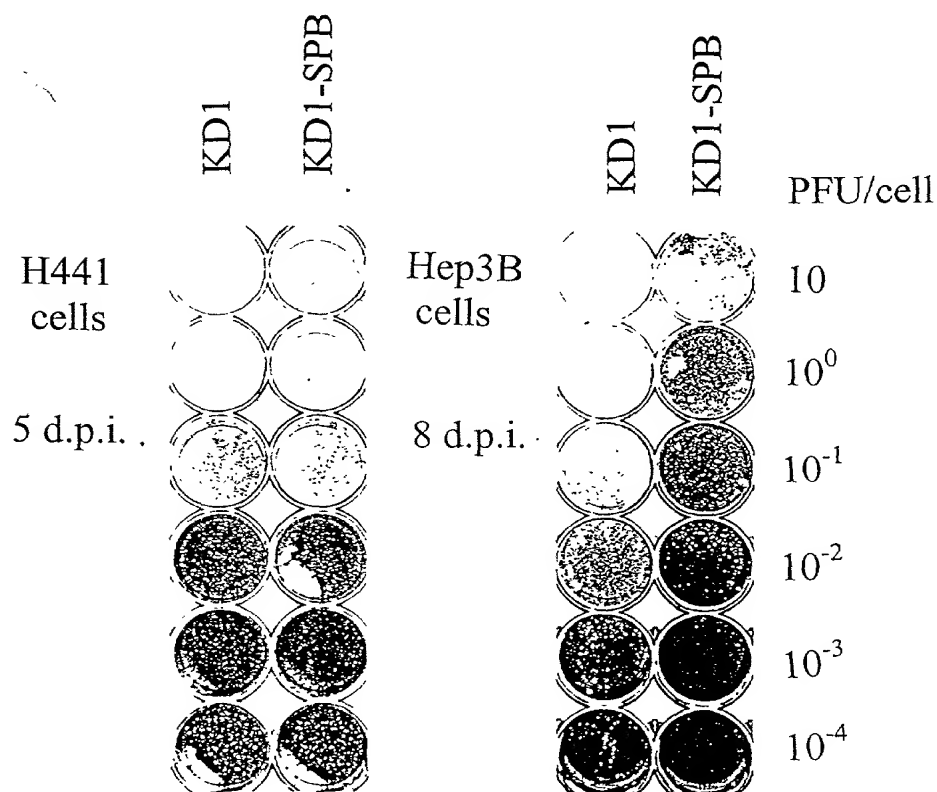


FIGURE 13

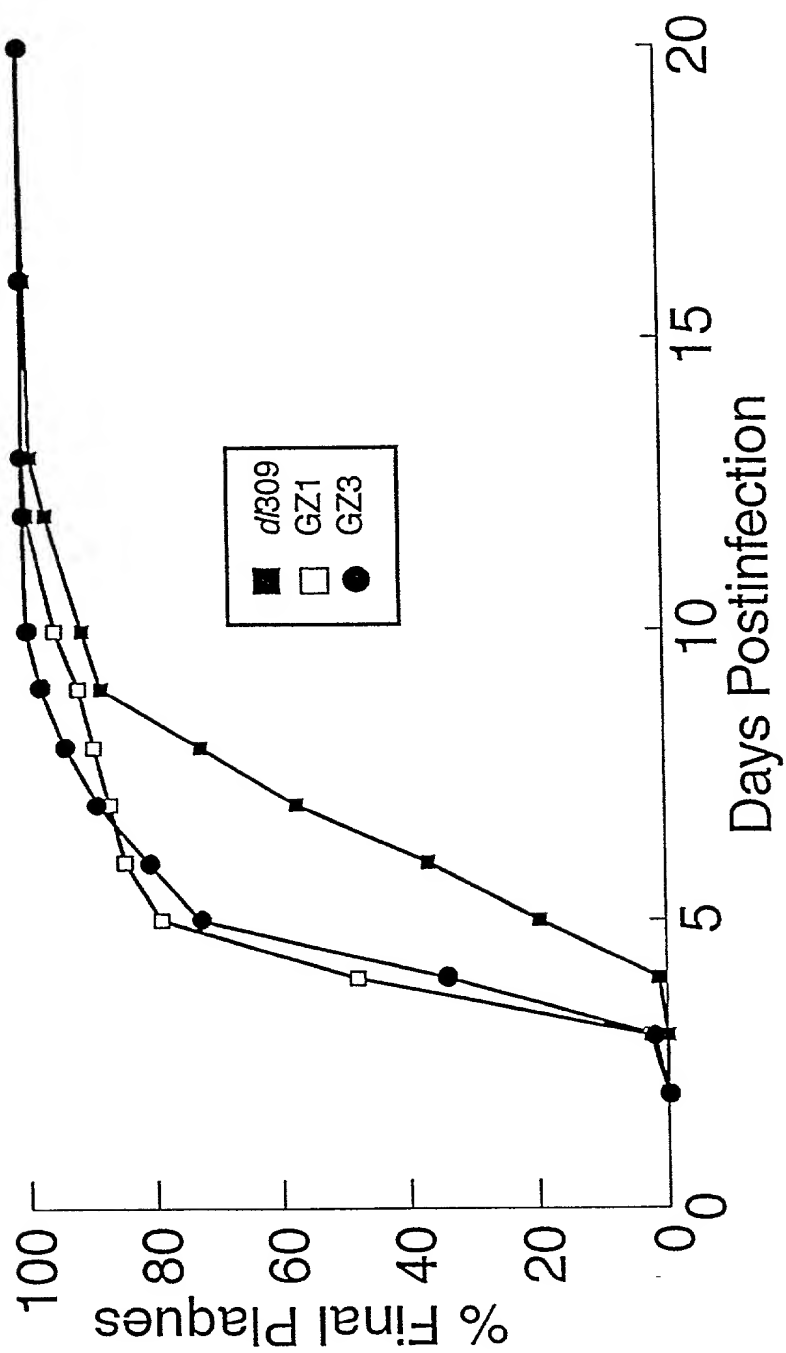


FIGURE 14

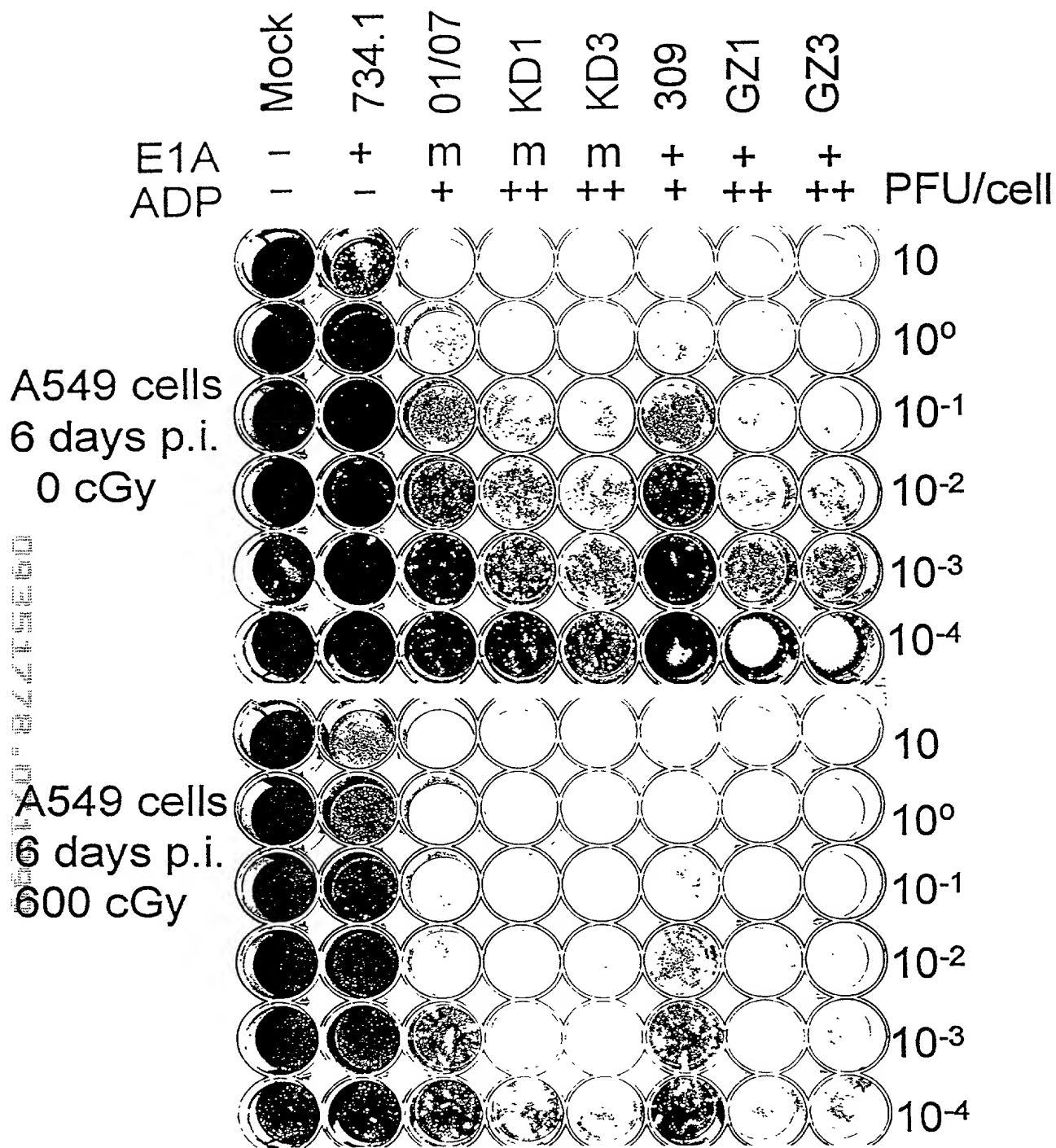


FIGURE 15

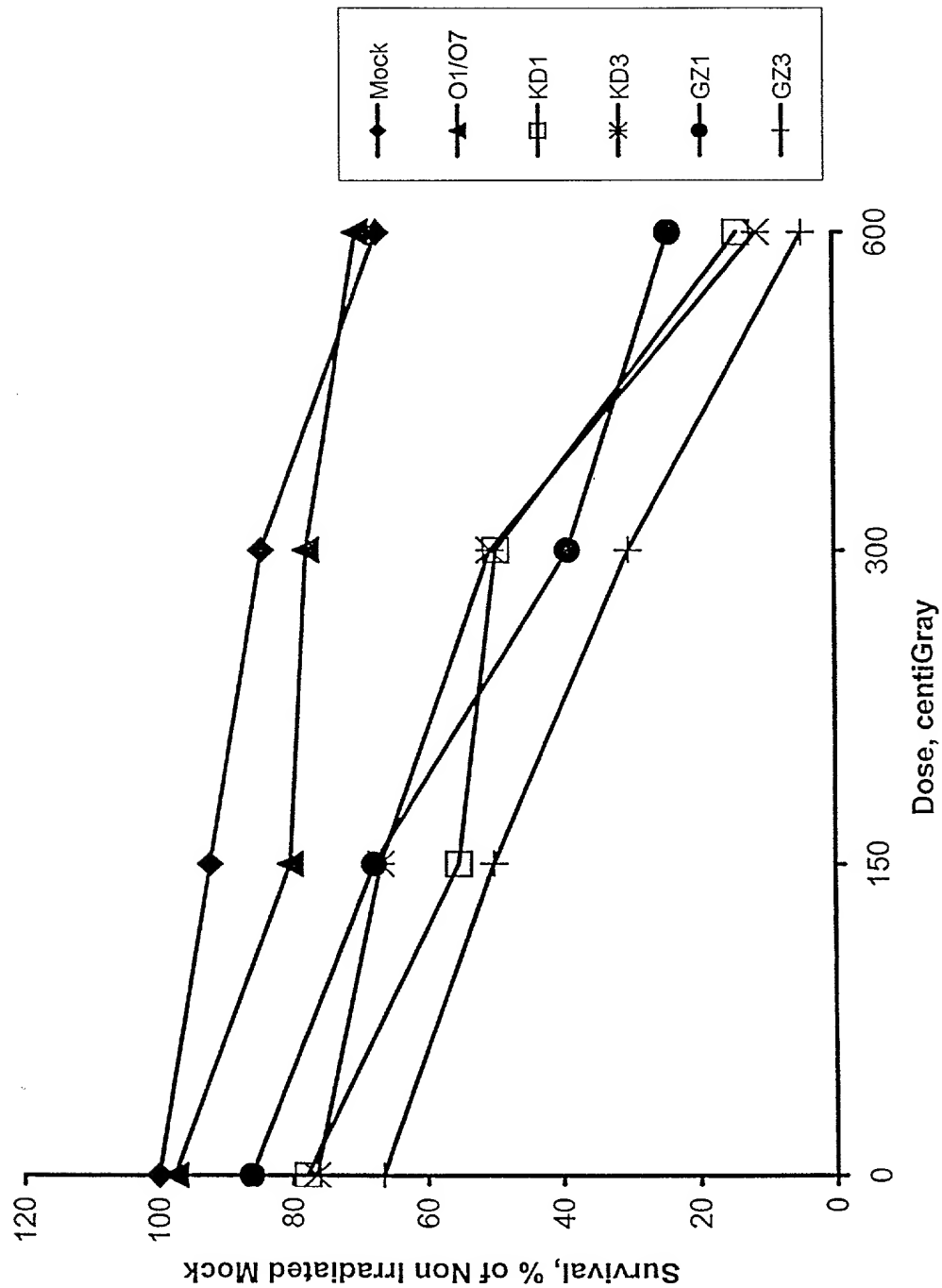


FIGURE 16

2024032600

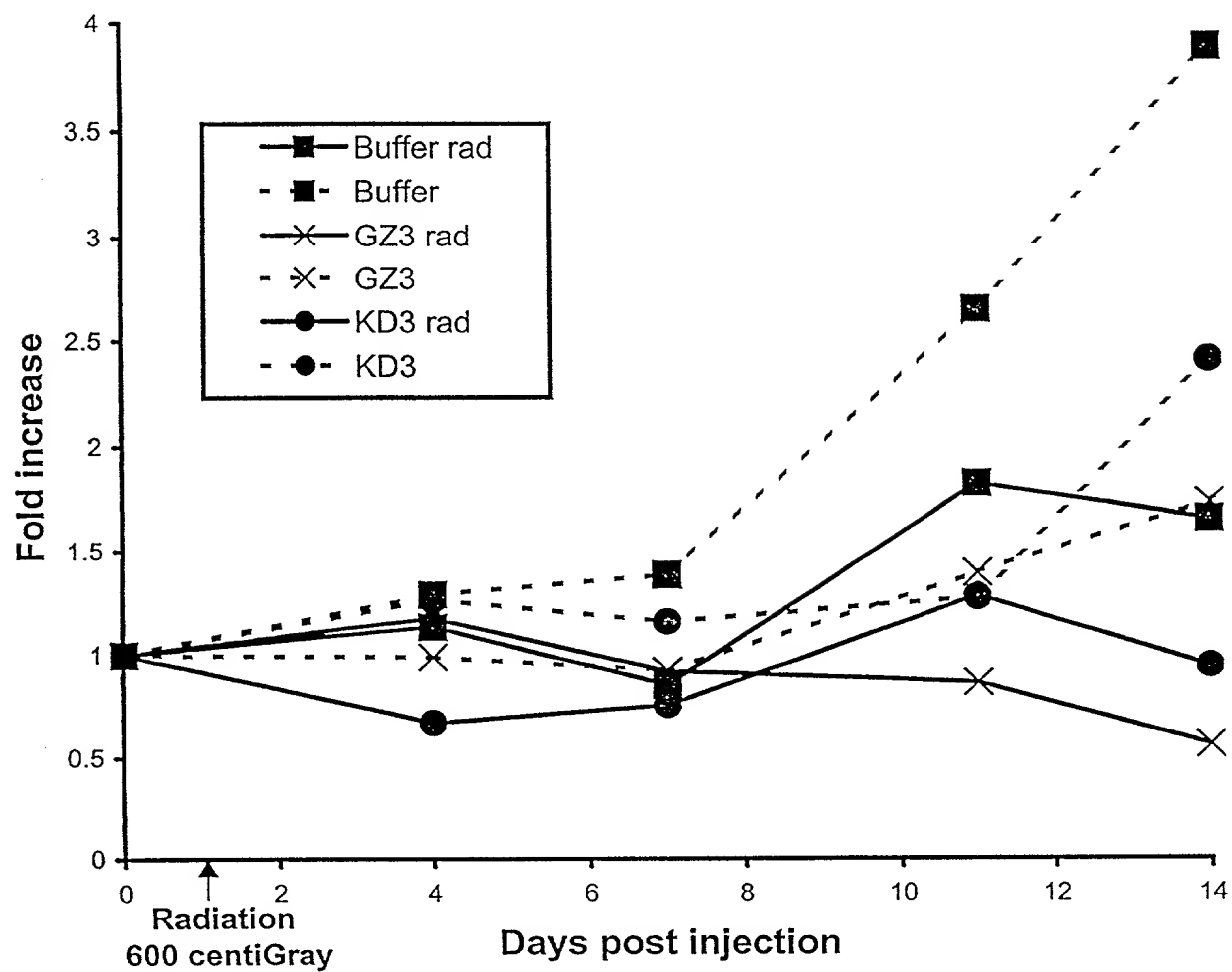


FIGURE 17

Ad2 Adenovirus Death Protein

Luminal Domain

MTGSTIAPTTDYRNTTATGLTSALNLPQVHAFVND 35

O - glycosylation *N - glycosylation*

WASLDMWWFSIALMFVGLIIMWLIGCLKRRRRARPP 70

*Transmembrane
(Signal - Anchor)*

Basic - Proline

IYRPIIVLNPHNEKIHRLDGLKPGSLLLQYD 101

Cytoplasmic - Nucleoplasmic Domain

FIGURE 18A

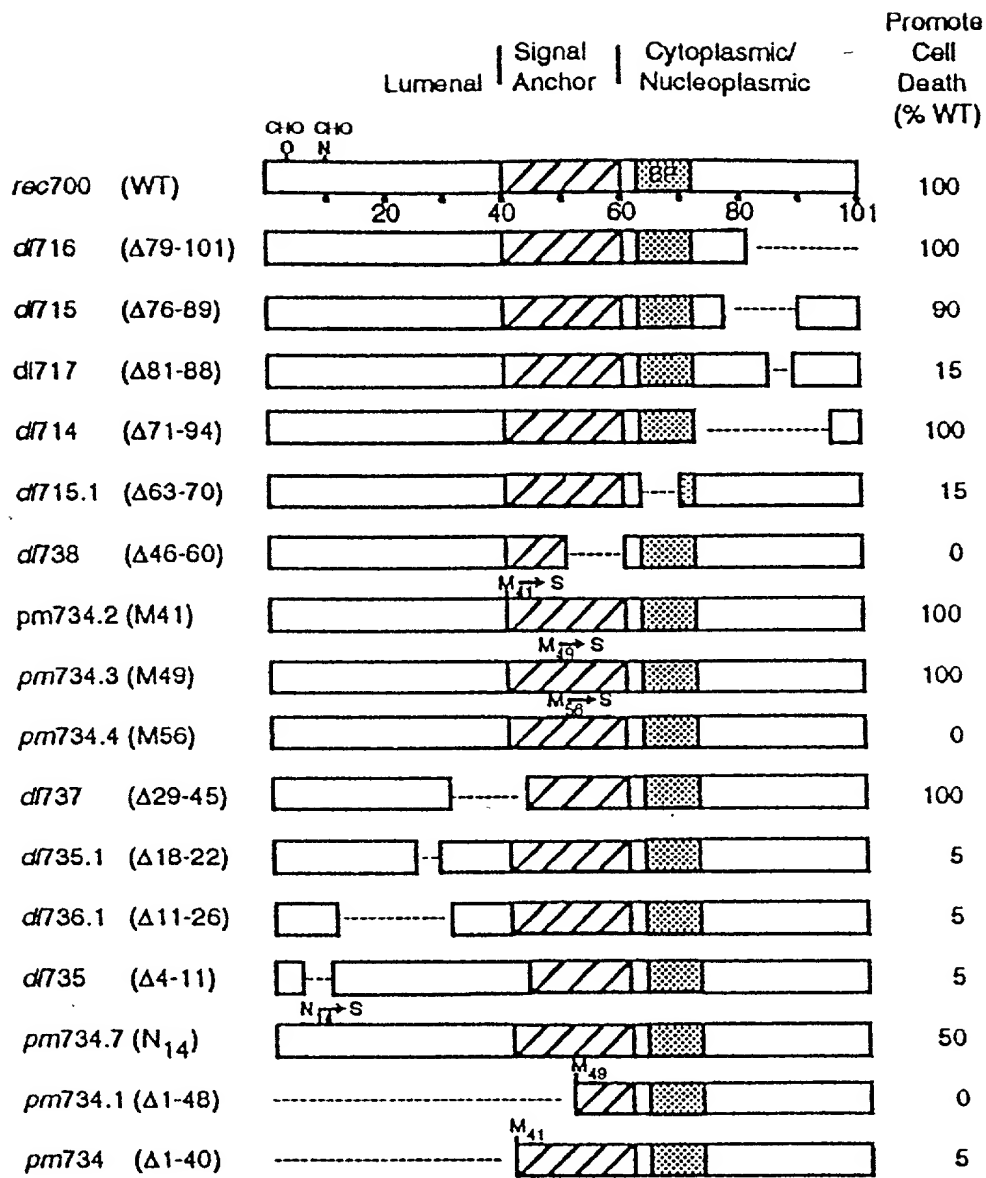


FIGURE 18B

662F40-8275660

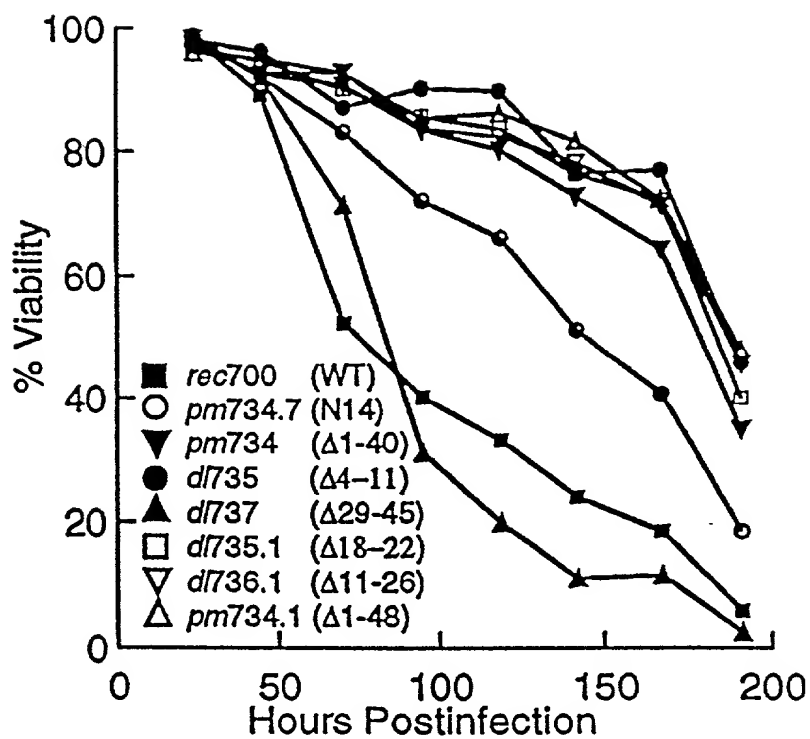


FIGURE 19A

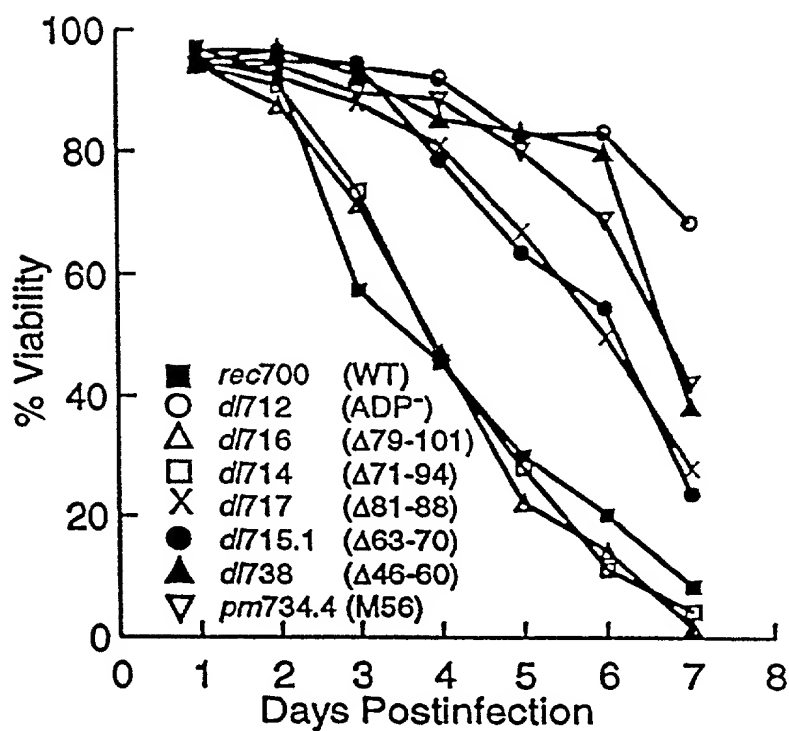


FIGURE 19B

Seq ID No.

		10	20	30	40	50
5	Ad1	-----MVD	T VNSYNTATGL	TSALNLPQVS	TFVNNWANLG	MWWFSIALMF
6	Ad2	MTGSTIAPTT	DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF
7	Ad5	-----MTN	TTNAAAATGL	TSTTNTQVS	AFVNNWDNLG	MWWFSIALMF
8	Ad6	-----MVD	T VNSYNTATGL	KSALNLPQVH	AFVNDWASLG	MWWFSIALMF
9	dl716	MTGSTIAPTT	DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF
10	dl715	MTGSTIAPTT	DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF
11	dl714	MTGSTIAPTT	DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF
12	dl737	MTGSTIAPTT	DYRNTTATGL	TSALNLPQ--	-----	-----IALMF

		60	70	80	90	100
5	Ad1	VCLIIMWLSC	CLKRKRARPP	IYKPIIVLNP	NNDGIHRLDG	LNTCSFSFAV -
6	Ad2	VCLIIMWLIC	CLKRRRARPP	IYRPIIVLNP	HNEKIHRLDG	LKPCSLLLQY D
7	Ad5	VCLIIMWLIC	CLKRKRARPP	IYSPIIVLHP	NNDGIHRLDG	LKHMFFSLTV -
8	Ad6	VCLIIMWLIC	CLKRRRARPP	IYRPIIVLNP	HNEKIHRLDG	LKPCSLLLQY D
9	dl716	VCLIIMWLIC	CLKRRRARPP	IYRPIIVL--	-----	-----
10	dl715	VCLIIMWLIC	CLKRRRARPP	IYRPI-----	-----G	LKPCSLLLQY D
11	dl714	VCLIIMWLIC	CLKRRRARPP	-----	-----	-----SLLLQY D
12	dl737	VCLIIMWLIC	CLKRRRARPP	IYRPIIVLNP	HNEKIHRLDG	LKPCSLLLQY D

Seq. ID No.

17	aa 1-40 of Ad2 ADP	MTGSTIAPTT DYRNTTATGL TSALNLPQVH AFVNDWASLD
18	aa 41-59 of Ad2 ADP	MWWFSIALMF VCLIIMWLI
19	aa 63-70 of Ad2 ADP	KRRRARPP
20	aa 60-101 of Ad2 ADP	C CLKRRRARPP IYRPIIVLNP HNEKIHRLDG LKPCSLLLQY D

FIGURE 20

LOCUS ad5 comple 35935 bp DNA SYN 06-FEB-1999
 DEFINITION ad5 complete genome
 ACCESSION ad5 comple
 KEYWORDS .
 SOURCE Unknown.
 ORGANISM Unknown
 Unclassified.
 REFERENCE 1 (bases 1 to 35935)
 AUTHORS Self
 JOURNAL Unpublished.
 BASE COUNT 8367 a 10073 c 9761 g 7734 t
 ORIGIN

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1 CATCATCAAT AATATACCTT ATTTTGGATT GAAGCCAATA TGATAATGAG GGGGTGGAGT
61 TTGTGACGTG GCGCGGGGCG TGGGAACGGG GCGGGTGACG TAGTAGTGTG GCGGAAGTGT
121 GATGTTGCAA GTGTGGCGGA ACACATGTAA GCGACGGATG TGGCAAAAGT GACGTTTTTG
181 GTGTGCGCCG GTGTACACAG GAAAGTGACAA TTTTCGCGCG GTTTTAGGCG GATGTTGTAG
241 TAAATTTGGG CGTAACCGAG TAAGATTTGG CCATTTTCGC GGGAAAAC TG AATAAGAGGA
301 AGTGAAATCT GAATAATTTT GTGTTACTCA TAGCGCGTAA TATTTGTCTA GGGCCGCGGG
361 GACTTTGACC GTTTACGTGG AGACTCGCCC AGGTGTTTTT CTCAGGTGTT TTCCGCGTTC
421 CGGGTCAAAG TTGGCGTTTT ATTATTATAG TCAGCTGACG TGTAAGTGTAT TTATACCCGG
481 TGAGTTCCTC AAGAGGCCAC TCTTGAGTGC CAGCGAGTAG AGTTTTCTCC TCCGAGCCGC
541 TCCGACACCG GGACTGAAAA TGAGACATAT TATCTGCCAC GGAGGTGTTA TTACCGAAGA
601 AATGGCCGCG AGTCTTTTGG ACCAGCTGAT CGAAGAGGTA CTGGCTGATA ATCTTCCACC
661 TCCTAGCCAT TTTGAACCAC CTACCCTTCA CGAACTGTAT GATTTAGACG TGACGGCCCC
721 CGAAGATCCC AACGAGGAGG CGGTTTCGCA GATTTTTCCC GACTCTGTAA TGTGGCGGT
781 GCAGGAAGGG ATTGACTTAC TCACTTTTCC GCCGGCGCCC GGTTCCTCCG AGCCGCTCA
841 CCTTCCCGG CAGCCCGAGC AGCCGGAGCA GAGAGCCTTG GGTCCGGTTT CTATGCCAAA
901 CCTTGTACCG GAGGTGATCG ATCTTACCTG CCACGAGGCT GGCTTTCAC CCAGTGACGA
961 CGAGGATGAA GAGGTGAGG AGTTTGTGTT AGATTATGTG GAGCACCCCG GGCACGGTTG
1021 CAGGTCTTGT CATTATCACC GGAGGAATAC GGGGGACCCA GATATTATGT GTTCGCTTTG
1081 CTATATGAGG ACCTGTGGCA TGTTTGTCTA CAGTAAGTGA AAATTATGGG CAGTGGGTGA
1141 TAGAGTGGTG GGTTTGGTGT GGTAAATTTT TTTTAAATTT TTACAGTTTT GTGTTTAAA
1201 GAATTTTGTA TTGTGATTTT TTAAAAAGGT CCTGTGTCTG AACCTGAGCC TGAGCCCGAG
1261 CCAGAACCGG AGCCTGCAAG ACCTACCCGC CGTCCTAAAA TGGCGCCTGC TATCCTGAGA
1321 CGCCCGACAT CACCTGTGTC TAGAGAATGC AATAGTAGTA CGGATAGCTG TGACTCCGGT
1381 CCTTCTAACA CACCTCCTGA GATACACCCG GTGGTCCCGC TGTGCCCAT TAAACCAAGT
1441 GCCGTGAGAG TTGGTGGGCG TCGCCAGGCT GTGGAATGTA TCGAGGACTT GCTTAACGAG
1501 CCTGGGCAAC CTTTGGACTT GAGCTGTAAA CGCCCCAGGC CATAAGGTGT AAACCTGTGA
1561 TTGCGTGTGT GGTAAACGCC TTTGTTTGCT GAATGAGTTG ATGTAAGTTT AATAAAGGGT
1621 GAGATAATGT TTAACCTGCA TGGCGTGTTA AATGGGGCGG GGCTTAAAGG GTATATAATG
1681 CGCCGTGGGC TAATCTTGGT TACATCTGAC CTCATGGAGG CTTGGGAGTG TTTGGAAGAT
1741 TTTTCTGCTG TCGCTAACTT GCTGGAACAG AGCTCTAACA GTACCTCTTG GTTTTGGAGG
1801 TTTCTGTGGG GCTCATCCCA GGCAAAGTTA GTCTGCAGAA TTAAGGAGGA TTACAAGTGG
1861 GAATTTGAAG AGCTTTTGAA ATCCTGTGGT GAGCTGTTTG ATTCTTTGAA TCTGGGTCAC
1921 CAGGCGCTTT TCCAAGAGAA GGTCAACAAG ACTTTGGATT TTTCCACACC GGGGCGCGCT
1981 GCGGCTGCTG TTGCTTTTTT GAGTTTATA AAGGATAAAT GGAGCGAAGA AACCCATCTG
2041 AGCGGGGGGT ACCTGCTGGA TTTTCTGGCC ATGCATCTGT GGAGAGCGGT TGTGAGACAC
2101 AAGAATCGCC TGCTACTGTT GTCTTCCGTC CGCCCGCGCA TAATACCGAG GGAGGAGCAG
2161 CAGCAGCAGC AGGAGGAAGC CAGGCGCGCG CGGCAGGAGC AGAGCCCATG GAACCCGAGA
2221 GCCGCGCTGG ACCCTCGGGA ATGAATGTTG TACAGGTGGC TGAAGTGTAT CCAGAACTGA
2281 GACGCATTTT GACAATTACA GAGGATGGGC AGGGGCTAAA GGGGGTAAAG AGGGAGCGGG
2341 GGGCTTGTGA GGCTACAGAG GAGGCTAGGA ATCTAGCTTT TAGCTTAATG ACCAGACACC
2401 GTCCTGAGTG TATTACTTTT CAACAGATCA AGGATAATTG CGCTAATGAG CTTGATCTGC
2461 TGGCGCAGAA GTATTCCATA GAGCAGCTGA CCACTTACTG GCTGCAGCCA GGGGATGATT
2521 TTGAGGAGGC TATTAGGGTA TATGCAAAGG TGGCACTTAG GCCAGATTGC AAGTACAAGA
2581 TCAGCAAATC TGTAAATATC AGGAATTGTT GCTACATTTT TGGGAACGGG GCCAGGTGG
2641 AGATAGATAC GGAGGATAGG GTGGCCTTTA GATGTAGCAT GATAAATATG TGGCCGGGGG

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2701 TGCTTGGCAT GGACGGGGTG GTTATTATGA ATGTAAGGTT TACTGGCCCC AATTTTAGCG
2761 GTACGGTTTT CCTGGCCAAT ACCAACCTTA TCCTACACGG TGTAAGCTTC TATGGGTTTA
2821 ACAATACCTG TGTGGAAGCC TGGACCGATG TAAGGGTTCG GGGCTGTGCC TTTTACTGCT
2881 GCTGGAAGGG GGTGGTGTGT CGCCCCAAAA GCAGGGCTTC AATTAAGAAA TGCCTCTTTG
2941 AAAGGTGTAC CTTGGGTATC CTGTCTGAGG GTAACCTCCAG GGTGCGCCAC AATGTGGCCT
3001 CCGACTGTGG TTGCTTCATG CTAGTGAAAA GCGTGGCTGT GATTAAGCAT AACATGGTAT
3061 GTGGCAACTG CGAGGACAGG GCCTCTCAGA TGCTGACCTG CTCGGACGGC AACTGTCACC
3121 TGCTGAAGAC CATTACGTA GCCAGCCACT CTCGCAAGGC CTGGCCAGTG TTTGAGCATA
3181 ACATACTGAC CCGCTGTTCC TTGCATTTGG GTAACAGGAG GGGGGTGTTC CTACCTTACC
3241 AATGCAATTT GAGTCACACT AAGATATTGC TTGAGCCCGA GAGCATGTCC AAGGTGAACC
3301 TGAACGGGGT GTTTGACATG ACCATGAAGA TCTGGAAGGT GCTGAGGTAC GATGAGACCC
3361 GCACCAGGTG CAGACCCTGC GAGTGTGGCG GTAAACATAT TAGGAACCAG CCTGTGATGC
3421 TGGATGTGAC CGAGGAGCTG AGGCCCCGAT ACTTGGTGTCT GGCCTGCACC CGCGCTGAGT
3481 TTGGCTCTAG CGATGAAGAT ACAGATTGAG GTACTGAAAT GTGTGGGCGT GGCTTAAGGG
3541 TGGGAAGAA TATATAAGGT GGGGGTCTTA TGTAAGTTTG TATCTGTTTT GCAGCAGCCG
3601 CCGCCGCCAT GAGCACCAC TCCTTTGATG GAAGCATTGT GAGCTCATAT TTGACAACGC
3661 GCATGCCCCC ATGGGCCGGG GTGCGTCAGA ATGTGATGGG CTCCAGCATT GATGGTCGCC
3721 CCGTCTGCC CGCAAACCTCT ACTACCTTGA CCTACGAGAC CGTGTCTGGA ACGCCGTTGG
3781 AGACTGCAGC CTCCGCCGCC GCTTCAGCCG CTGCAGCCAC CGCCCGCGGG ATTGTGACTG
3841 ACTTTGCTTT CCTGAGCCCG CTTGCAAGCA GTGCAGCTTC CCGTTCATCC GCCCGCGATG
3901 ACAAGTTGAC GGCTCTTTTG GCACAATTGG ATTCTTTGAC CCGGGAACCTT AATGCTGTTT
3961 CTCAGCAGCT GTTGGATCTG CGCCAGCAGG TTTCTGCCCT GAAGGCTTCC TCCCCTCCCA
4021 ATGCGGTTTA AAACATAAAT AAAAAACAG ACTCTGTTTG GATTTGGATC AAGCAAGTGT
4081 CTTGTGTCTT TTATTTAGGG GTTTTGC GCGG TAGGC CCGGGACCAG CCGTCTCGGT
4141 CGTTGAGGGT CCGTGTGATT TTTTCCAGGA CGTGGTAAAG GTGACTCTGG ATGTTTCAGAT
4201 ACATGGGCAT AAGCCCGTCT CTGGGGTGGA GGTAGCACCA CTGCAGAGCT TCATGCTGCG
4261 GGGTGGTGTT GTAGATGATC CAGTCGTAGC AGGAGCGCTG GCGGTGGTGC CTAAGAAATGT
4321 CTTTCAGTAG CAAGCTGATT GCCAGGGGCA GGCCCTTGGT GTAAGTGTTT ACAAGCGGT
4381 TAAGCTGGGA TGGGTGCATA CGTGGGGATA TGAGATGCAT CTTGGACTGT ATTTTLAGGT
4441 TGGCTATGTT CCCAGCCATA TCCCTCCGGG GATTCATGTT GTGCAGAACC ACCAGCACAG
4501 TGTATCCGGT GCACTTGCGA AATTTGTCAT GTAGCTTAGA AGGAAATGCG TGGAAAGAACT
4561 TGGAGACGCC CTTGTGACCT CCAAGATTTT CCATGCATTC GTCCATAATG ATGGCAATGG
4621 GCCACGGGG GCGGCGCTGG GCGAAGATAT TTCTGGGATC ACTAACGTCA TAGTTGTGTT
4681 CCAGGATGAG ATCGTCATAG GCCATTTTFA CAAAGCGCGG GCGGAGGGTG CCAGACTGCG
4741 GTATAATGGT TCCATCCGGC CCAGGGGCGT AGTTACCTC ACAGATTGTC ATTTCCACG
4801 CTTTGAGTTC AGATGGGGGG ATCATGTCTA CCTGCGGGG GATGAAGAAA ACGGTTTCCG
4861 GGGTAGGGGA GATCAGCTGG GAAGAAAGCA GGTTCCTGAG CAGCTGCGAC TTACCGCAGC
4921 CGGTGGGCCC GTAAATCACA CCTATTACCG GGTGCAACTG GTAGTTAAGA GAGCTGCAGC
4981 TGCCGTCATC CTTGAGCAGG GGGGCCACTT CGTTAAGCAT GTCCCTGACT CGCATGTTTT
5041 CCCTGACCAA ATCCGCCAGA AGGCGCTCGC CGCCAGCGA TAGCAGTTCT TCGAAGGAAAG
5101 CAAAGTTTTT CAACGGTTTG AGACCGTCCG CCGTAGGCAT GCTTTTGAGC GTTTGACCAA
5161 GCAGTTCCAG GCGGTCCCAC AGCTCGGTCA CCTGCTCTAC GGCATCTCGA TCCAGCATAT
5221 CTCCTCGTTT CGCGGGTTGG GCGGCTTTC GCTGTACGGC AGTAGTCGGT GCTCGTCCAG
5281 ACGGGCCAGG GTCATGTCTT TCCACGGGCG CAGGGTCCTC GTCAGCGTAG TCTGGGTACAC
5341 GGTGAAGGGG TCGCTCCGG GCTGCGCGCT GGCCAGGGTG CGCTTGAGGC TGGTCCTGCT
5401 GGTGCTGAAG CGCTGCCGGT CTTGCGCCCTG CGCGTCGGCC AGGTAGCATT TGACCATGGT
5461 GTCATAGTCC AGCCCCCTCG CGGCGTGGCC CTTGGCGCGC AGCTTGCCCT TGGAGGAGGC
5521 GCCGCACGAG GGGCAGTGCA GACTTTTGAG GGCGTAGAGC TTGGGCGCGA GAAATACCGA
5581 TTCCGGGGG TAGGCATCCG CGCCGACAGC CCCGACAGC GTCTCGCATT CCACGAGCCA
5641 GGTGAGCTCT GGCCGTTTCG GGTCAAAAAC CAGGTTTCCC CCATGCTTTT TGATGCGTTT
5701 CTTACCTCTG GTTTCCATGA GCCGGTGTCC ACGCTCGGTG ACGAAAAGGC TGTCCGTGTC
5761 CCCGTATACA GACTTGAGAG GCCTGTCTCT GAGCGGTGTT CCGCGGTCCT CCTCGTATAG
5821 AAACCTCGGAC CACTCTGAGA CAAAGGCTCG CGTCCAGGCC AGCACGAAGG AGGCTAAGTG
5881 GGAGGGGTAG CGGTCGTTGT CCACTAGGGG GTCCACTCGC TCCAGGGTGT GAAGACACAT
5941 GTCGCCCTCT TCGGCATCAA GGAAGGTGAT TGGTTTGTAG GTGTAGGCCA CGTGACCGGG
6001 TGTTCTTGAA GGGGGGCTAT AAAAGGGGGT GGGGGCGCGT TCGTCTCAC TCTCTCCGC
6061 ATCGCTGTCT GCGAGGGCCA GCTGTTGGGG TGAGTACTCC CTCTGAAAAG CGGGCATGAC

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FIGURE 21
(SHEET 2)

6121	TTCTGCGCTA	AGATTGTCAG	TTTCCAAAAA	CGAGGAGGAT	TTGATATTCA	CCTGGCCCCG
6181	GGTGAGCCCT	TTGAGGGTGG	CCGCATCCAT	CTGGTCAGAA	AAGACAATCT	TTTTGTGTGC
6241	AAGCTTGGTG	GCAAACGACC	CGTAGAGGGC	GTTGGACAGC	AACTTGCGCA	TGGAGCGCAG
6301	GGTTTGGTTT	TTGTGCGGAT	CGGCGCGCTC	CTTGCCGCG	ATGTTTAGCT	GCACGTATTC
6361	GCGCGCAACG	CACCGCCATT	CGGAAAAGAC	GGTGGTGCGC	TCGTGCGGCA	CCAGGTGCAC
6421	GCGCCAACCG	CGGTTGTGCA	GGGTGACAAG	GTCAACGCTG	GTGGCTACCT	CTCCGCGTAG
6481	GCGCTCGTTG	GTCCAGCAGA	GGCGGCCGCC	CTTGCGCGAG	CAGAATGGCG	GTAGGGGGTC
6541	TAGCTGCGTC	TCGTCCGGGG	GGTCTGCGTC	CACGGTAAAG	ACCCCGGGCA	GCAGCGCGC
6601	GTCGAAGTAG	TCTATCTTGC	ATCCTTGCAA	GTCTAGCGCC	TGCTGCCATG	CGCGGGCGCG
6661	AAGCGCGCGC	TCGTATGGGT	TGAGTGGGGG	ACCCCATGGC	ATGGGGTGGG	TGAGCGCGGA
6721	GGCGTACATG	CCGCAAATGT	CGTAAACGTA	GAGGGGCTCT	CTGAGTATTC	CAAGATATGT
6781	AGGGTAGCAT	CTTCCACCGC	GGATGCTGGC	GCGCACGTAA	TCGTATAGTT	CGTGCGAGGG
6841	AGCGAGGAGG	TCGGGACCGA	GGTTGCTACG	GGCGGGCTGC	TCTGCTCGGA	AGACTATCTG
6901	CCTGAAGATG	GCATGTGAGT	TGGATGATAT	GGTTGGACGC	TGGAAGACGT	TGAAGCTGGC
6961	GTCTGTGAGA	CCTACCGCGT	CACGCACGAA	GGAGGCGTAG	GAGTCGCGCA	GC'TTGT'TGAC
7021	CAGCTCGGCG	GTGACCTGCA	CGTCTAGGGC	GCAGTAGTCC	AGGGT'TT'CCT	TGATGATGTC
7081	ATACTTATCC	TGTCCTTTTT	TTTTCCACAG	CTCGCGGTTG	AGGACAAACT	CTTCGCGGTC
7141	TTTCCAGTAC	TCTTGGATCG	GAAACCCGTC	GGCCTCCGAA	CGGTAAAGAC	CTAGCATGTA
7201	GAAGTGGTTG	ACGGCCTGGT	AGGCGCAGCA	TCCCTTTTCT	ACGGGTAGCG	CGTATGCCTG
7261	CGCGGCCTTC	CGGAGCGGAG	TGTGGGTGAG	CGCAAAGGTG	TCCCTGACCA	TGACTTTGAG
7321	GTACTGGTAT	TTGAAGTCAG	TGTGCTCGCA	TCCGCCCTGC	TCCCAGAGCA	AAAAGTCCGT
7381	GCGCTTTTTG	GAACGCGGAT	TTGGCAGGGC	GAAGGTGACA	TCGTTGAAGA	GTATCTTTCC
7441	CGCGCGAGGC	ATAAAGTTGC	GTGTGATGCG	GAAGGGTCCC	GGCACCTCGG	AACGGTTGTT
7501	AATTACCTGG	GCGGCGAGCA	CGATCTCGTC	AAAGCCGTTG	ATGTTGTGGC	CCACAATGTA
7561	AAGTTCCAAG	AAGCGCGGGA	TGCCCTTGAT	GGAAGGCAAT	TTTTTAAGTT	CCTCGTAGGT
7621	GAGCTCTTCA	GGGGAGCTGA	GCCCCGTGCTC	TGAAAGGGCC	CAGTCTGCAA	GATGAGGGTT
7681	GGAAGCGACG	AATGAGCTCC	ACAGGTCACG	GGCCATTAGC	ATTTGACAGT	GGTCGCGAAA
7741	GGTCCTAAAC	TGGCGACCTA	TGGCCATTTT	TTCTGGGGTG	ATGCAGTAGA	AGGTAAGCGG
7801	GTCTTGTTC	CAGCGGTCCC	ATCCAAGGTT	CGCGGCTAGG	TTCGCGCGG	CAGTCACTAG
7861	AGGCTCATCT	CCGCGGAACT	TCATGACGAG	CATGAAGGGC	ACGAGCTGCT	TCCCAAAGGC
7921	CCCCATCAA	GATATAGTCT	CTACATCGTA	GGTGACAAAG	AGACGCTCGG	TGCGAGGATG
7981	CGAGCCGATC	GGGAAGAACT	GGATCTCCCG	CCACCAATTG	GAGGAGTGGC	TATTGATGTG
8041	GTGAAAAGTAG	AAGTCCCTGC	GACGGGCCGA	ACACTCGTGC	TGGCTTTTGT	AAAAACGTGC
8101	GCAGTACTGG	CAGCGGTGCA	CGGGCTGTAC	ATCCTGCACG	AGGTTGACCT	GACGACCGCG
8161	CACAAGGAAG	CAGAGTGGGA	ATTTGAGCCC	CTCGCCTGGC	GGGTTTGGCT	GGTGGTCTTC
8221	TACTTCGCT	GCTTGTCTTT	GACCGTCTGG	CTGCTCGAGC	GGAGTTACGG	TGGATCGGAC
8281	CACCACGCCG	CGCGAGCCCA	AAGTCCAGAT	GTCCCGCGCG	GGCGGTGCGA	GCTTGATGAC
8341	AACATCCGCG	AGATGGGAGC	TGTCCATGGT	CTGGAGCTCC	CGCGGCGTCA	GGTCAGGCGG
8401	GAGCTCTGTC	AGGTTTAACT	CGCATAGACG	GGTCAGGGCG	CGGGCTAGAT	CCAGGTGATA
8461	CCTAATTTTC	AGGGGCTGGT	TGGTGCGCGC	GTGATGGCT	TGCAAGAGGC	CGCATCCCCG
8521	CGGCGCGACT	ACGGTACCGC	GCGGCGGGCG	GTGGGCCGCG	GGGGTGTCTT	TGGATGATGC
8581	ATCTAAAAGC	GGTGACGCGG	GCGAGCCCCC	GGAGGTAGGG	GGGGCTCCGG	ACCCGCCGGG
8641	AGAGGGGGCA	GGGGCACGTC	GGCGCCGCCG	GCGGGCAGGA	GCTGGTGCTG	CGCGCGTAGG
8701	TTGCTGGCGA	ACGCGACGAC	GCGGCGGTTG	ATCTCCTGAA	TCTGGCGCCT	CTGCGTGAAG
8761	ACGACGGGCC	CGGTGAGCTT	GAGCCTGAAA	GAGAGTTGCA	CAGAATCAAT	TTCCGTGTCTG
8821	TTGACGGCGG	CCTGGCGCAA	AATCTCTTGC	AGTCTCCTG	AGTTGTCTTG	ATAGGCGATC
8881	TCGGCCATGA	ACTGCTCGAT	CTCTTCTTCC	TGGAGATCTC	CGCGTCCGGC	TCGCTCCACG
8941	GCGGCGGCGA	GGTCGTTTGA	AATGCGGGCC	ATGAGCTGCG	AGAAGGCGTT	GAGGCTTCCC
9001	TCGTTCCAGA	CGCGGCTGTA	GACCACGCCC	CCTTCGGCAT	CGCGGGCGCG	CATGACCACC
9061	TGCGCGAGAT	TGAGCTCCAC	GTGCCGGGCG	AAGACGGCGT	AGTTTCGCAG	GCGCTGAAAG
9121	AGGTAGTTGA	GGGTGGTGGC	GGTGTGTTCT	GCCACGAAGA	AGTACATAAC	CCAGAGTCCG
9181	AACGTGGATT	CGTTGATATC	CCCCAAGGCC	TCAAGGCGCT	CCATGGCCCTC	GTAGACGTCC
9241	ACGGCGAAGT	TGAAAAACTG	GGAGTTGCGC	GCCGACACGG	TTAATCTCTC	CTCCAGAAGA
9301	CGGATGAGCT	CGGCGACAGT	GTCGCGCACG	TCGCGCTCAA	AGGCTACAGG	GGCCTCTTCT
9361	TCTTCTTCAA	TCTCCTCTTC	CATAAGGGCC	TCCCCTTCTT	CTTCTTCTGG	CGGCGGTGGG
9421	GGAGGGGGGA	CAC				

ad5

9541	AGTTGGAAGA	CGCCGCCCGT	CATGTCCCGG	TTATGGGTTG	GCGGGGGGCT	GCCATGCGGC
9601	AGGGATACGG	CGCTAACGAT	GCATCTCAAC	AATTGTTGTG	TAGGTACTCC	GCCGCCGAGG
9661	GACCTGAGCG	AGTCCGCATC	GACCGGATCG	GAAAACCTCT	CGAGAAAGGC	GTCTAACCG
9721	TCACAGTCGC	AAGGTAGGCT	GAGCACCGTG	GCGGGCGGCA	GCGGGCGGCG	GTGGGGGTTG
9781	TTTCTGGCGG	AGGTGCTGCT	GATGATGTAA	TTAAAGTAGG	CGGTCTTGAG	ACGGCGGATG
9841	GTCGACAGAA	GCACCATGTC	CTTGGGTCCG	GCCTGCTGAA	TGCGCAGGCG	GTGGGCCATG
9901	CCCCAGGCTT	CGTTTTGACA	TCGGCGCAGG	TCTTTGTAGT	AGTCTTGTCAT	GAGCCTTTCT
9961	ACCGGCACTT	CTTCTTCTCC	TTCTCTTGT	CCTGCATCTC	TTGCATCTAT	CGCTGCGGCG
10021	GCGGCGGAGT	TTGGCCGTAG	GTGGCGCCCT	CTTCTCTCCA	TGCGTGTGAC	CCCGAAGCCC
10081	CTCATCGGCT	GAAGCAGGGC	TAGGTGCGCG	ACAACGCGCT	CGGCTAATAT	GGCCTGCTGC
10141	ACCTGCGTGA	GGGTAGACTG	GAAGTCATCC	ATGTCCACAA	AGCGGTGGTA	TGCGCCCGTG
10201	TTGATGGTGT	AAGTGCAGTT	GGCCATAACG	GACCAGTTAA	CGGTCTGGTG	ACCCGGCTGC
10261	GAGAGCTCGG	TGTACCTGAG	ACGCGAGTAA	GCCCTCGAGT	CAAATACGTA	GTGCTTGCAA
10321	GTCCGCACCA	GGTACTGGTA	TCCCACCAAA	AAGTGCGGCG	GCGGCTGGCG	GTAGAGGGGC
10381	CAGCGTAGGG	TGGCCGGGCG	TCCGGGGGCG	AGATCTTCCA	ACATAAGGCG	ATGATATCCG
10441	TAGATGTACC	TGGACATCCA	GGTGATGCCG	GCGGCGGTGG	TGGAGGCGCG	CGGAAAGTCG
10501	CGGACGCGGT	TCCAGATGTT	GCGCAGCGGC	AAAAAGTGCT	CCATGGTCGG	GACGCTCTGG
10561	CCGGTCAGGC	GCGCGCAATC	GTTGACGCTC	TAGACCGTGC	AAAAGGAGAG	CCTGTAAGCG
10621	GGCACTCTTC	CGTGGTCTGG	TGGATAAATT	CGCAAGGGTA	TCATGGCGGA	CGACCGGGGT
10681	TCGAGCCCCG	TATCCGCGCG	TCCGCGGTGA	TCCATGCGGT	TACCGCCCCG	GTGTGCAACC
10741	CAGGTGTGCG	ACGTCAGACA	ACGGGGGAGT	GCTCCTTTTG	GCTTCCTTCC	AGGCGCGGCG
10801	GCTGCTGCGC	TAGCTTTTTT	GGCCACTGGC	CGCGCGCAGC	GTAAGCGGTT	AGGCTGGAAA
10861	GCGAAAGCAT	TAAGTGGCCT	GCTCCCTGTA	GCCGGAGGGT	TATTTTCCAA	GGGTGTGATC
10921	GCGGGACCCC	CGGTTCCAGT	CTCGGACCGG	CCGGATGCG	GCGAACGGGG	GTGTGCTTCC
10981	CCGTGATGCA	AGACCCCGCT	TGCAAAATTCC	TCCGGAACA	GGGACGAGCC	CCTTTTTTGC
11041	TTTTCCAGAG	TGCATCCGGT	GCTGCGGCAG	ATGCGCCCCC	CTCCTCAGCA	GCGGCAAGAG
11101	CAAGAGCAGC	GGCAGACATG	CAGGGCACCC	TCCCCTCCTC	CTACCGCGTC	AGGAGGGGCG
11161	ACATCCGCGG	TTGACGCGGC	AGCAGATGGT	GATTACGAAC	CCCCGCGGCG	CCGGGCCCCG
11221	CACTACCTGG	ACTTGGAGGA	GGGCGAGGGC	CTGGCGCGGC	TAGGAGCGCC	CTCTCCTGAG
11281	CGGTACCCAA	GGGTGCAGCT	GAAGCGTGAT	ACGCGTGAGG	CGTACGTGCC	GCGGCAGAAC
11341	CTGTTTCGCG	ACCGCGAGGG	AGAGGAGCCC	GAGGAGATGC	GGGATCGAAA	GTTCACGCA
11401	GCGCGCGAGC	TGCGGCATGG	CCTGAATCGC	GAGCGGTTGC	TGCGCGAGGA	GGACTTTGAG
11461	CCCGACGCGC	GAACCGGGAT	TAGTCCCGCG	CGCGCACACG	TGGCGGCGCG	CGACCTGGTA
11521	ACCGCATACG	AGCAGACGGT	GAACCGAGAG	ATTAACCTTC	AAAAAAGCTT	TAACAACCAC
11581	GTGCGTACGC	TTGTGGCGCG	CGAGGAGGTG	GCTATAGGAC	TGATGCATCT	GTGGGACTTT
11641	GTAAGCGCGC	TGGAGCAAAA	CCCAAATAGC	AAGCCGCTCA	TGGCGCAGCT	GTCTCTTATA
11701	GTGCAGCACA	GCAGGGACAA	CGAGGCATTC	AGGGATGCGC	TGCTAAACAT	AGTAGAGCCC
11761	GAGGGCCGCT	GGCTGCTCGA	TTTGATAAAC	ATCCTGCAGA	GCATAGTGGT	GCAGGAGCGC
11821	AGCTTGAGCC	TGGCTGACAA	GGTGGCCGCC	ATCAACTATT	CCATGCTTAG	CCTGGGCAAG
11881	TTTTACGCC	GCAAGATATA	CCATACCCCT	TACGTTCCCA	TAGACAAGGA	GGTAAAGATC
11941	GAGGGGTTCT	ACATGCGCAT	GGCGCTGAAG	GTGCTTACCT	TGAGCGACGA	CCTGGGCGTT
12001	TATCGCAACG	AGCGCATCCA	CAAGGCCGTG	AGCGTGAGCC	GGCGGCGCGA	GCTCAGCGAC
12061	CGCGAGCTGA	TGCACAGCCT	GCAAAGGGCC	CTGGCTGGCA	CGGGCAGCGG	CGATAGAGAG
12121	GCCGAGTCCT	ACTTTGACGC	GGGCGCTGAC	CTGCGCTGGG	CCCCAAGCCG	ACGCGCCCTG
12181	GAGGCAGCTG	GGGCCGGACC	TGGGCTGGCG	GTGGCACCCG	CGCGCGCTGG	CAACGTCCGG
12241	GGCGTGAGAG	AATATGACGA	GGACGATGAG	TACGAGCCAG	AGGACGGCGA	GTAATAAGCG
12301	GTGATGTTTC	TGATCAGATG	ATGCAAGACG	CAACGGACCC	GGCGGTGCGG	GCGGCGCTGC
12361	AGAGCCAGCG	GTCCGGCCTT	AACTCCACGG	ACGACTGGCG	CCAGGTCATG	GACCGCATCA
12421	TGTCGCTGAC	TGCGCGCAAT	CCTGACGCGT	TCCGGCAGCA	GCCGCGAGCC	AACCGGCTCT
12481	CCGCAATTCT	GGAAGCGGTG	GTCCCGGCGC	GCGCAAACCC	CACGCACGAG	AAGGTGCTGG
12541	CGATCGTAAA	CGCGCTGGCC	GAAAACAGGG	CCATCCGGCC	CGACGAGGCC	GGCCTGGTCT
12601	ACGACGCGCT	GCTTCAGCGC	GTGGCTCGTT	ACAACAGCGG	CAACGTGCAG	ACCAACCTGG
12661	ACCGGCTGGT	GGGGGATGTG	CGCGAGGCCG	TGGCGCAGCG	TGAGCGCGCG	CAGCAGCAGG
12721	GCAACCTGGG	CTCCATGGTT	GCACTAAACG	CCTTCCTGAG	TACACAGCCC	GCCAACGTGC
12781	CGCGGGGACA	GGAGGACTAC	ACCAACTTTG	TGAGCGCACT	GCGGCTAATG	GTGACTGAGA
12841	CACCGCAAAG	TGAGGTGTAC	CAGTCTGGGC	CAGACTATTT	TTTCCAGACC	AGTAGACAAG
12901	GCCTGCAGAC	CGTAAACCTG	AGCCAGGCTT	TCAAAAACTT	GCAGGGGCTG	TGGGGGGTGC

FIGURE 21
(SHEET 4)


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19801 GCCCAACAGG CCTAATTACA TTGCTTTTAG GGACAATTTT ATTGGTCTAA TGTATTACAA
19861 CAGCACGGGT AATATGGGTG TTCTGGCGGG CCAAGCATCG CAGTTGAATG CTGTTGTAGA
19921 TTTGCAAGAC AGAAACACAG AGCTTTCATA CCAGCTTTTG CTTGATTCCA TTGGTGATAG
19981 AACCAGGTAC TTTTCTATGT GGAATCAGGC TGTTGACAGC TATGATCCAG ATGTTAGAAT
20041 TATTGAAAAT CATGGAAGT AAGATGAAT TCCAAATTAC TGCTTTCCAC TGGGAGGTGT
20101 GATTAATACA GAGACTCTTA CCAAGGTAAA ACCTAAAACA GGTGAGGAAA ATGGATGGGA
20161 AAAAGATGCT ACAGAATTTT CAGATAAAAA TGAAATAAGA GTTGGAATAA ATTTTGCCAT
20221 GGAAATCAAT CTAAATGCCA ACCTGTGGAG AAATTTCTTG TACTCCAACA TAGCGCTGTA
20281 TTTGCCCCGAC AAGCTAAAGT ACAGTCCTTC CAACGTAAAA ATTTCTGATA ACCCAAACAC
20341 CTACGACTAC ATGAACAAGC GAGTGGTGGC TCCCGGGTTA GTGGACTGCT ACATTAACCT
20401 TGGAGCACGC TGGTCCCTTG ACTATATGGA CAACGTCAAC CCATTTAACC ACCACCGCAA
20461 TGCTGGCCTG CGCTACCGCT CAATGTTGCT GGGCAATGGT CGCTATGTGC CTTCCACAT
20521 CCAGGTGCCT CAGAAGTTCT TTGCCATTAA AAACCTCCTT CTCTGCCGG GCTCATACAC
20581 CTACGAGTGG AACTTCAGGA AGGATGTTAA CATGGTTCTG CAGAGCTCCC TAGGAAATGA
20641 CCTAAGGTTT GACGGAGCCA GCATTAAGTT TGATAGCATT TGCTTTTACG CCACCTTCTT
20701 CCCCATGGCC CACAACACCG CCTCCACGCT TGAGGCCATG CTTAGAAACG ACACCAACGA
20761 CCAGTCCTTT AACGACTATC TCTCCGCCGC CAACATGCTC TACCCTATAC CCGCCAACGC
20821 TACCAACGTG CCCATATCCA TCCCCTCCCG CAACTGGGCG GCTTTCCGCG GCTGGGCCTT
20881 CACGCGCCTT AAGACTAAGG AAACCCCATC ACTGGGCTCG GGCTACGACC CTTATTACAC
20941 CTACTCTGGC TCTATACCCT ACCTAGATGG AACCTTTTAC CTCAACCACA CTTTAAAGAA
21001 GGTGGCCATT ACCTTTGACT CTTCTGTGAG CTGGCCTGGC AATGACCGCC TGCTTACCCC
21061 CAACGAGTTT GAAATTAAGC GCTCAGTTGA CGGGGAGGGT TACAACGTTG CCCAGTGTAA
21121 CATGACCAA GACTGGTTCC TGGTACAAAT GCTAGCTAAC TACAACATTG GCTACCAGGG
21181 CTTCTATATC CCAGAGAGCT ACAAGGACCG CATGTACTCC TTCTTTAGAA ACTTCCAGCC
21241 CATGAGCGCT CAGGTGGTGG ATGATACTAA ATACAAGGAC TACCAACAGG TGGGCATCCT
21301 ACACCAACAC AACAACTCTG GATTTGTTGG CTACCTTGCC CCCACCATGC GCGAAGGACA
21361 GGCCTACCCT GCTAACTTCC CCTATCCGCT TATAGGCAAG ACCGCAGTTG ACAGCATTAC
21421 CCAGAAAAAG TTTCTTTGCG ATCGCACCTT TTGGCGCATC CCATTCTCCA GTAACTTTAT
21481 GTCCATGGGC GCACTCACAG ACCTGGGCCA AAACCTTCTC TACGCCAACT CCGCCCACGC
21541 GCTAGACATG ACTTTTGAGG TGGATCCCAT GGACGAGCCC ACCCTTCTTT ATGTTTTGTT
21601 TGAAGTCTTT GACGTGGTCC GTGTGCACCG GCCGCACCGC GGCGTCATCG AAACCGTGTA
21661 CCTGCGCAGC CCCTTCTCGG CCGGCAACCG CACAACATAA AGAAGCAAGC AACATCAACA
21721 ACAGCTGCCG CCATGGGCTC CAGTGAGCAG GAACTGAAAG CCATTGTCAA AGATCTTGGT
21781 TGTGGGCCAT ATTTTTTGGG CACCTATGAC AAGCGCTTTC CAGGCTTTGT TTCTCCACAC
21841 AAGCTCGCCT GCGCCATAGT CAATACGGCC GGTGCGGAGA CTGGGGGCGT ACACTGGATG
21901 GCCTTTGCCT GGAACCCGCA CTCAAAAACA TGCTACCTCT TTGAGCCCTT TGGCTTTTCT
21961 GACCAGCGAC TCAAGCAGGT TTACCAGTTT GAGTACGAGT CACTCCTGCG CCGTAGCGCC
22021 ATTGCTTCTT CCCCCGACCG CTGTATAACG CTGGAAGAGT CCACCCAAAG CGTACAGGGG
22081 CCCAACTCGG CCGCCTGTGG ACTATTCTGC TGCATGTTTC TCCACGCCTT TGCCAACTGG
22141 CCCCAACTC CCATGGATCA CAACCCACCC ATGAACCTTA TTACCGGGGT ACCCAACTCC
22201 ATGCTCAACA GTCCCCAGGT ACAGCCACCC CTGCGTCGCA ACCAGGAACA GCTCTACAGC
22261 TTCTTGAGC GCCACTCGCC CTACTTCCGC AGCCACAGTG CGCAGATTAG GAGCGCCACT
22321 TCTTTTGTG ACTTGAAAAA CATGTAAAAA TAATGTACTA GAGACACTTT CAATAAAGGC
22381 AAATGCTTTT ATTTGTACAC TCTCGGTTGA TTATTTACCC CCACCCTTGC CGTCTGCGCC
22441 GTTTAAAAAT CAAAGGGGTT CTGCCGCGCA TCGCTATGCG CCACTGGCAG GGACACGTTG
22501 CGATACTGGT GTTTAGTGCT CCACTTAAAC TCAGGCACAA CCATCCGCGG CAGCTCGGTG
22561 AAGTTTTTAC TCCACAGGCT GCGCACCATC ACCAACGCGT TTAGCAGGTC GGGCGCCGAT
22621 ATCTTGAAGT CGCAGTTGGG GCCTCCGCCC TGCGCGCGCG AGTTGCGATA CACAGGGTTG
22681 CAGCACTGGA ACACATACAG CGCCGGGTGG TGCACGCTGG CCAGCACGCT CTTGTGCGAG
22741 ATCAGATCCG CGTCCAGGTC CTCCGCGTTG CTCAGGGCGA ACGGAGTCAA CTTTGGTAGC
22801 TGCCTTCCCA AAAAGGGCGC GTGCCCAGGC TTTGAGTTGC ACTCGCACCG TAGTGGCATC
22861 AAAAGGTGAC CGTGCCCGGT CTGGGCGTTA GGATACAGCG CCTGCATAAA AGCCTTGATC
22921 TGCTTAAAAG CCACCTGAGC CTTTGCGCCT TCAGAGAAGA ACATGCCGCA AGACTTGCCG
22981 GAAAAC TGAT TGGCCGGACA GGCCGCGTCG TGCACGCAGC ACCTTGCGTC GGTGTTGGAG
23041 ATCTGCACCA CATTTGCGCC CCACCGGTTT TTCACGATCT TGGCCTTGCT AGACTGCTCC
23101 TTCAGCGCGC GTGCCCCTT TCGCTCGTC ACATCCATT CAATCACGT CTCCTTATTT
23161 ATCATAATGC TTCCGTGTAG ACACCTAAGC TCGCCTTCGA TCTCAGCGCA GCGGTGACAG

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FIGURE 21
(SHEET 7)

23221	CACAACGCGC	AGCCCGTGGG	CTCGTGATGC	TTGTAGGTCA	CCTCTGCAAA	CGACTGCAGG
23281	TACGCCTGCA	GGAATCGCCC	CATCATCGTC	ACAAAGGTCT	TGTTGCTGGT	GAAGGTCAGC
23341	TGCAACCCGC	GGTGCTCCTC	GTTTCAGCCAG	GTCTTGCCATA	CGGCCGCCAG	AGCTTCCACT
23401	TGGTCAGGCA	GTAGTTTGAA	GTTTCGCTTT	AGATCGTTAT	CCACGTGGTA	CTTGTCCTATC
23461	AGCGCGCGCG	CAGCCTCCAT	GCCCTTCTCC	CACGCAGACA	CGATCGGCAC	ACTCAGCGGG
23521	TTCATCACCG	TAATTTCACT	TTCCGCTTCG	CTGGGCTCTT	CCTCTTCTCT	TTGCGTCCGC
23581	ATACCACGCG	CCACTGGGTC	GTCTTCATTG	AGCCGCCGCA	CTGTGCGCTT	ACCTCTTTTG
23641	CCATGCTTGA	TTAGCACCAG	TGGGTTGCTG	AAACCCACCA	TTTGTAGCGC	CACATCTTCT
23701	CTTTCTTCTT	CGCTGTCCAC	GATTACCTCT	GGTGATGGCG	GGCGCTCGGG	CTTGGGAGAA
23761	GGGCGCTTCT	TTTTCTTCTT	GGGCGCAATG	GCCAAATCCG	CCGCCGAGGT	CGATGGCCGC
23821	GGGCTGGGTG	TGCGCGGCAC	CAGCGCGTCT	TGTGATGAGT	CTTCCTCGTC	CTCGGACTCG
23881	ATACGCCGCC	TCATCCGCTT	TTTTGGGGGG	GCCCGGGGAG	GCGGCGGCGA	CGGGGACGGG
23941	GACGACACGT	CCTCCATGGT	TGGGGGACGT	CGCGCCGCAC	CGCGTCCGCG	CTCGGGGGTG
24001	GTTTCGCGCT	GCTCCTCTTC	CCGACTGGCC	ATTTCTTCTT	CCTATAGGCA	GAAAAAGATC
24061	ATGGAGTCAG	TCGAGAAGAA	GGACAGCCTA	ACCGCCCCCT	CTGAGTTTCG	CACCACCGCC
24121	TCCACCGATG	CCGCCAACGC	GCCTACCACC	TTCCCCGTCG	AGGCACCCCC	GCTTGAGGAG
24181	GAGGAAGTGA	TTATCGAGCA	GGACCCAGGT	TTTGTAAGCG	AAGACGACGA	GGACCGCTCA
24241	GTACCAACAG	AGGATAAAAA	GCAAGACCAG	GACAACGCAG	AGGCAAACGA	GGAACAAGTC
24301	GGGCGGGGGG	ACGAAAGGCA	TGGCGACTAC	CTAGATGTGG	GAGACGACGT	GCTGTTGAAG
24361	CATCTGCAGC	GCCAGTGC GC	CATTATCTGC	GACGCGTTGC	AAGAGCGCAG	CGATGTGCCC
24421	CTCGCCATAG	CGGATGTCTG	CCTTGCCCTA	GAACGCCACC	TATTCTCACC	GCGCGTACCC
24481	CCCAAACGCC	AAGAAAACGG	CACATGCGAG	CCCAACCCGC	GCCTCAACTT	CTACCCCGTA
24541	TTTGCCGTGC	CAGAGGTGCT	TGCCACCTAT	CACATCTTTT	TCCAAAACCT	CAAGATAACC
24601	CTATCCTGCC	GTGCCAACCG	CAGCGAGCGC	GACAAGCAGC	TGGCCTTGCG	GCAGGGCGCT
24661	GTACATCTTG	ATATCGCCTC	GCTCAACGAA	GTGCCAAAAA	TCTTTGAGGG	TCTTGACGCG
24721	GACGAGAAGC	GCGCGGCAAA	CGCTCTGCAA	CAGGAAAACA	GCGAAAATGA	AAGTCACTCT
24781	GGAGTGTTGG	TGGAACCTCG	GGGTGACAAC	GCGCGCCTAG	CCGTACTAAA	ACGCAGCATC
24841	GAGGTCACCC	ACTTTGCCTA	CCCGGCACTT	AACCTACCCC	CCAAGGTCAT	GAGCACAGTC
24901	ATGAGTGAGC	TGATCGTGCG	CCGTGCGCAG	CCCCTGGAGA	GGGATGCAAA	TTTGCAAGAA
24961	CAAACAGAGG	AGGGCCTACC	CGCAGTTGGC	GACGAGCAGC	TAGCGCGCTG	GCTTCAAACG
25021	CGCGAGCCTG	CCGACTTGGA	GGAGCGACGC	AAACTAATGA	TGGCCGCAGT	GCTCGTTACC
25081	GTGGAGCTTG	AGTGCATGCA	GCGGTTCTTT	GCTGACCCGG	AGATGCAGCG	CAAGCTAGAG
25141	GAAACATTGC	ACTACACCTT	TCGACAGGGT	TACGTACGCC	AGGCCTGCAA	GATCTCCAAC
25201	GTGGAGCTCT	GCAACCTGGT	CTCCTACCTT	GGAATTTTGC	ACGAAAACCG	CCTTGGGCAA
25261	AACGTGCTTC	ATTCCACGCT	CAAGGGCGAG	GCGCGCCGCG	ACTACGTCCG	CGACTGCGTT
25321	TACTTATTTT	TATGCTACAC	CTGGCAGACG	GCCATGGGCG	TTTGGCAGCA	GTGCTTGAG
25381	GAGTGCAACC	TCAAGGAGCT	GCAGAACTG	CTAAAGCAAA	ACTTGAAGGA	CCTATGGACG
25441	GCCTTCAACG	AGCGCTCCGT	GGCCGCGCAC	CTGGCGGACA	TCATTTTCCC	CGAACGCCCTG
25501	CTTAAAACCC	TGCAACAGGG	TCTGCCAGAC	TTCAACAGTC	AAAGCATGTT	GCAGAACTTT
25561	AGGAACTTTA	TCCTAGAGCG	CTCAGGAATC	TTGCCCGCCA	CCTGCTGTGC	ACTTCTTAGC
25621	GACTTTGTGC	CCATTAAGTA	CCGCGAATGC	CCTCCGCCGC	TTTGGGGCCA	CTGCTACCTT
25681	CTGCAGCTAG	CCAACCTACC	TGCCTACCAC	TCTGACATAA	TGGAAGACGT	GAGCGGTGAC
25741	GGTCTACTGG	AGTGTCACTG	TCGCTGCAAC	CTATGCACCC	CGCACCGCTC	CCTGGTTTGC
25801	AATTCGCAGC	TGCTTAACGA	AAGTCAAATT	ATCGGTACCT	TTGAGCTGCA	GGGTCCCTCG
25861	CCTGACGAAA	AGTCCGCGGC	TCCGGGGTTG	AAACTCACTC	CGGGGCTGTG	GACGTCGGCT
25921	TACCTTCGCA	AATTTGTACC	TGAGGACTAC	CACGCCCACG	AGATTAGGTT	CTACGAAGAC
25981	CAATCCCGCC	CGCCAAATGC	GGAGCTTACC	GCCTGCGTCA	TTACCCAGGG	CCACATTCTT
26041	GGCCAATTGC	AAGCCATCAA	CAAAGCCCGC	CAAGAGTTTC	TGCTACGAAA	GGGACGGGGG
26101	GTTTACTTGG	ACCCCGAGTC	CGGCGAGGAG	CTCAACCCAA	TCCCCCGGCC	GCCGCAGCCC
26161	TATCAGCAGC	AGCCGCGGGC	CCTTGCTTCC	CAGGATGGCA	CCCCAAAAGA	AGCTGCAGCT
26221	GCCGCCGCCA	CCCACGGACG	AGGAGGAATA	CTGGGACAGT	CAGGCAGAGG	AGGTTTTTGA
26281	CGAGGAGGAG	GAGGACATGA	TGGAAGACTG	GGAGAGCCTA	GACGAGGAAG	CTTCCGAGGT
26341	CGAAGAGGTG	TCAGACGAAA	CACCGTCACC	CTCGGTCGCA	TTCCCCCTCG	CGGCGCCCCA
26401	GAAATCGGCA	ACCGGTTCCA	GCATGGCTAC	AACCTCCGCT	CCTCAGGCGC	CGCCGGCACT
26461	GCCCGTTTCG	CGACCCAACC	GTAGATGGGA	CACCACTGGA	ACCAGGGCCG	GTAAGTCCAA
26521	GCAGCCGCCG	CCGTTAGCCC	AAGAGCAACA	ACAGCGCCAA	GGCTACCGCT	CATGGCGCGG
26581	GCACAAGAAC	GCCATAGTTG	CTTGCTTGCA	AGACTGTGGG	GGCAACATCT	CCTTCGCCCC

26641	CCGCTTTTCTT	CTCTACCATC	ACGGCGTGGC	CTTCCCCCGT	AACATCCTGC	ATTACTACCG
26701	TCATCTCTAC	AGCCCATACT	GCACCGGCGG	CAGCGGCAGC	GGCAGCAACA	GCAGCGGCCA
26761	CACAGAAGCA	AAGGCGACCG	GATAGCAAGA	CTCTGACAAA	GCCCAAGAAA	TCCACAGCGG
26821	CGGCAGCAGC	AGGAGGAGGA	GCGCTGCGTC	TGGCGCCCAA	CGAACCCGTA	TCGACCCGCG
26881	AGCTTAGAAA	CAGGATTTTT	CCCCTCTGT	ATGCTATATT	TCAACAGAGC	AGGGGCCAAG
26941	AACAAGAGCT	AAAAATAAAA	AACAGGTCTC	TGCGATCCCT	CACCCGCAGC	TGCCTGTATC
27001	ACAAAAGCGA	AGATCAGCTT	CGGCGCACGC	TGGAAGACGC	GGAGGCTCTC	TTCAGTAAAT
27061	ACTGCGCGCT	GACTCTTAAG	GACTAGTTTC	GCGCCCTTTC	TCAAATTTAA	GCGCGAAAAC
27121	TACGTCATCT	CCAGCGGCCA	CACCCGGCGC	CAGCACCTGT	CGTCAGCGCC	ATTATGAGCA
27181	AGGAAATTCC	CACGCCCTAC	ATGTGGAGTT	ACCAGCCACA	AATGGGACTT	GCGGCTGGAG
27241	CTGCCCAAGA	CTACTCAACC	CGAATAAACT	ACATGAGCGC	GGGACCCAC	ATGATATCCC
27301	GGGTCAACGG	AATCCGCGCC	CACCGAAACC	GAATTCTCTT	GGAACAGGCG	GCTATTACCA
27361	CCACACCTCG	TAATAACCTT	AATCCCCGTA	GTTGGCCCCG	TGCCCTGGTG	TACCAGGAAA
27421	GTCCCGCTCC	CACCACTGTG	GTACTTCCCA	GAGACGCCCA	GGCCGAAGTT	CAGATGACTA
27481	ACTCAGGGG	GCAGCTTGCG	GGCGGCTTTC	GTACAGGGT	GCGGTCGCCC	GGGCAGGGTA
27541	TAACTCACCT	GACAATCAGA	GGCGAGGTA	TTCAGCTCAA	CGACGAGTCG	GTGAGCTCCT
27601	CGCTTGCTCT	CCGTCCGGAC	GGGACATTTT	AGATCGGCGG	CGCCGGCCGT	CCTTCATTCA
27661	CGCCTCGTCA	GGCAATCCTA	ACTCTGCAGA	CCTCGTCCTC	TGAGCCGCGC	TCTGGAGGCA
27721	TTGGAACCTCT	GCAATTTATT	GAGGAGTTTG	TGCCATCGGT	CTACTTTAAC	CCCTTCTCGG
27781	GACCTCCCGG	CCACTATCCG	GATCAATTTA	TTCCTAACTT	TGACGCGGTA	AAGGACTCGG
27841	CGGACGGCTA	CGACTGAATG	TTAAGTGGAG	AGGCAGAGCA	ACTGCGCCTG	AAACACCTGG
27901	TCCACTGTCT	CCGCCACAAG	TGCTTTGCC	GCGACTCCGG	TGAGTTTTCG	TACTTTGAAT
27961	TGCCCCGAGGA	TCATATCGAG	GGCCCGGCGC	ACGGCGTCCG	GCTTACCGCC	CAGGGAGAGC
28021	TTGCCCGTAG	CCTGATTCGG	GAGTTTACCC	AGCGCCCCCT	GCTAGTTGAG	CGGGACAGGG
28081	GACCCGTGTG	TCTCACTGTG	ATTTGCAACT	GTCCCTAACCT	TGGATTACAT	CAAGATCTTT
28141	GTTGCCATCT	CTGTGCTGAG	TATAATAAAT	ACAGAAATTA	AAATATACTG	GGGCTCCTAT
28201	CGCCATCCTG	TAAACGCCAC	CGTCTTCACC	CGCCCAAGCA	AACCAAGGCG	AACCTTACCT
28261	GGTACTTTTA	ACATCTCTCC	CTCTGTGATT	TACAACAGTT	TCAACCCAGA	CGGAGTGAGT
28321	CTACGAGAGA	ACCTCTCCGA	GCTCAGCTAC	TCCATCAGAA	AAAACACCAC	CCTCCTTACC
28381	TGCCGGGAAC	GTACGAGTGC	GTCACCGGCC	GCTGCACCAC	ACCTACCGCC	TGACCGTAAA
28441	CCAGACTTTT	TCCGGACAGA	CCTCAATAAC	TCTGTTTACC	AGAACAGGAG	GTGAGCTTAG
28501	AAAACCCTTA	GGGTATTAGG	CCAAAGGCGC	AGCTACTGTG	GGGTTTATGA	ACAATTCAAG
28561	CAACTCTACG	GGCTATTCTA	ATTCAGGTTT	CTCTAGAATC	GGGGTTGGGG	TTATTCTCTG
28621	TCTTGCTGATT	CTCTTTATTC	TTATACTAAC	GCTTCTCTGC	CTAAGGCTCG	CCGCCTGCTG
28681	TGTGCACATT	TGCATTTATT	GTCAGCTTTT	TAAACGCTGG	GGTCGCCACC	CAAGATGATT
28741	AGGTACATAA	TCCTAGGTTT	ACTCACCTTT	GCGTCAGCCC	ACGGTACCAC	CCAAAAGGTG
28801	GATTTTAAAG	AGCCAGCCTG	TAATGTTACA	TTCGCAGCTG	AAGCTAATGA	GTGCACCACT
28861	CTTATAAAAT	GCACCACAGA	ACATGAAAAG	CTGCTTATTC	GCCACAAAAA	CAAAATTGGC
28921	AAGTATGCTG	TTTATGCTAT	TTGGCAGCCA	GGTGACACTA	CAGAGTATAA	TGTTACAGTT
28981	TTCCAGGGTA	AAAGTCATAA	AACTTTTATG	TATACTTTTC	CATTTTATGA	AATGTGCGAC
29041	ATTACCATGT	ACATGAGCAA	ACAGTATAAG	TTGTGGCCCC	CACAAAATTG	TGTGGAAAAC
29101	ACTGGCACTT	TCTGCTGCAC	TGCTATGCTA	ATTACAGTGC	TCGCTTTGGT	CTGTACCCTA
29161	CTCTATATTA	AATACAAAAG	CAGACGCAGC	TTTATTTGAGG	AAAAGAAAAT	GCCTTAATTT
29221	ACTAAGTTAC	AAAGCTAATG	TCACCACTAA	CTGCTTTACT	CGCTGCTTGC	AAAACAAATT
29281	CAAAAAGTTA	GCATTATAAT	TAGAATAGGA	TTTAAACCCC	CCGGTCATTT	CCTGCTCAAT
29341	ACCATTCCCC	TGAACAATTG	ACTCTATGTG	GGATATGCTC	CAGCGCTACA	ACCTTGAAGT
29401	CAGGCTTCCT	GGATGTCAGC	ATCTGACTTT	GGCCAGCACC	TGTCCCGCGG	ATTTGTTCCA
29461	GTCCAACCTAC	AGCGACCCAC	CCTAACAGAG	ATGACCAACA	CAACCAACGC	GGCCGCGGCT
29521	ACCGGACTTA	CATCTACCAC	AAATACACCC	CAAGTTTCTG	CCTTTGTCAA	TAAGTGGGAT
29581	AACTTGCGCA	TGTGGTGGTT	CTCCATAGCG	CTTATGTTTG	TATGCCTTAT	TATTATGTGG
29641	CTCATCTGCT	GCCTAAAGCG	CAAACGCGCC	CGACCACCCA	TCTATAGTCC	CATCATTGTG
29701	CTACACCCAA	ACAATGATGG	AATCCATAGA	TTGGACGGAC	TGAAACACAT	GTTCTTTTCT
29761	CTTACAGTAT	GATTAAATGA	GACATGATTC	CTCGAGTTT	TATATTACTG	ACCCTTGTTG
29821	CGCTTTTCTT	TGCGTGCTCC	ACATTGGCTG	CGGTTTCTCA	CATCGAAGTA	GACTGCATTC
29881	CAGCCTTCAC	AGTCTATTTG	CTTTACGGAT	TTGTCACCC	CACGCTCATC	TGCAGCCTCA
29941	TCACTGTGGT	CATCGCCTTT	ATCCAGTGCA	TTGACTGGGT	CTGTGTGCGC	TTTGCATATC
30001	TCAGACACCA	TCCCCAGTAC	AGGGACAGGA	CTATAGCTGA	GCTTCTTAGA	ATTCTTTAAT

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30061 TATGAAATTT ACTGTGACTT TTCTGCTGAT TATTTGCACC CTATCTGCGT TTTGTTCCCC
30121 GACCTCCAAG CCTCAAAGAC ATATATCATG CAGATTCACT CGTATATGGA ATATTCCAAG
30181 TTGCTACAAT GAAAAAAGCG ATCTTTCCGA AGCCTGGTTA TATGCAATCA TCTCTGTTAT
30241 GGTGTTCTGC AGTACCATCT TAGCCCTAGC TATATATCCC TACCTTGACA TTGGCTGGAA
30301 ACGAATAGAT GCCATGAACC ACCCAACTTT CCCCAGCGCC GCTATGCTTC CACTGCAACA
30361 AGTTGTTGCC GCGCGCTTTG TCCCAGCCAA TCAGCCTCGC CCCACTTCTC CCACCCCCAC
30421 TGAAATCAGC TACTTTAATC TAACAGGAGG AGATGACTGA CACCCTAGAT CTAGAAATGG
30481 ACGGAATTAT TACAGAGCAG CGCCTGCTAG AAAGACGCAG GGCAGCGGCC GAGCAACAGC
30541 GCATGAATCA AGAGCTCCAA GACATGGTTA ACTTGCACCA GTGCAAAAGG GGTATCTTTT
30601 GTCTGGTAAA GCAGGCCAAA GTCACCTACG ACAGTAATAC CACCGGACAC CGCCTTAGCT
30661 ACAAGTTGCC AACCAAGCGT CAGAAATTGG TGGTCATGGT GGGAGAAAAG CCCATTACCA
30721 TAACTCAGCA CTCGGTAGAA ACCGAAGGCT GCATTCACTC ACCTTGTCAG GGACCTGAGG
30781 ATCTCTGCAC CCTTATTAAG ACCCTGTGCG GTCTCAAAGA TCTTATTCCC TTTAACTAAT
30841 AAAAAAAGAT AATAAAGCAT CACTTACTTA AAATCAGTTA GCAAATTTCT GTCCAGTTTA
30901 TTCAGCAGCA CCTCCTTGCC CTCCTCCCAG CTCTGGTATT GCAGCTTCCT CCTGGCTGCA
30961 AACTTTCTCC ACAATCTAAA TGGAATGTCA GTTTCCTCCT GTTCCTGTCC ATCCGCACCC
31021 ACTATCTTCA TGTTGTTGCA GATGAAGCGC GCAAGACCGT CTGAAGATAC CTTCAACCCC
31081 GTGTATCCAT ATGACACGGA AACCAGTCTT CCAACTGTGC CTTTCTTAC TCCTCCCTTT
31141 GTATCCCCCA ATGGGTTTCA AGAGAGTCCC CCTGGGGTAC TCTCTTTGCG CCTATCCGAA
31201 CCTCTAGTTA CCTCCAATGG CATGCTTGCG CTCAAAATGG GCAACGGCCT CTCTCTGGAC
31261 GAGGCCGGCA ACCTTACCTC CAAAATGTGA ACCACTGTGA GCCACCTCT CAAAAAACC
31321 AAGTCAAACA TAAACCTGGA AATATCTGCA CCCCTCACAG TTACCTCAGA AGCCCTAAGT
31381 GTGGCTGCCG CCGCACCTCT AATGGTCGCG GCAACACAC TCACCATGCA ATCACAGGCC
31441 CCGCTAACCG TGCACGACTC CAAACTTAGC ATTGCCACCC AAGGACCCCT CACAGTGTCA
31501 GAAGGAAAGC TAGCCCTGCA AACATCAGGC CCCCTCACCA CCACCGATAG CAGTACCCTT
31561 ACTATCACTG CCTCACCCCC TCTAACTACT GCCACTGGTA GCTTGGGCAT TGACTTGAAA
31621 GAGCCCATTT ATACACAAAA TGGAAACTA GGACTAAAGT ACGGGGCTCC TTTGCATGTA
31681 ACAGACGACC TAAACACTTT GACCGTAGCA ACTGGTCCAG GTGTGACTAT TAATAATACT
31741 TCCTTGCAAA CTAAAGTTAC TGGAGCCTTG GGTTTTGATT CACAAGGCAA TATGCAACTT
31801 AATGTAGCAG GAGGACTAAG GATTGATTCT CAAAACAGAC GCCTTATACT TGATGTTAGT
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 ORGANISM Unknown
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BASE COUNT 7744 a 9470 c 9285 g 7093 t

ORIGIN

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 61 TTGTGACGTG GCGCGGGGCG TGGGAACGGG GCGGGTGACG TAGTAGTGTG GCGGAAGTGT
 121 GATGTTGCAA GTGTGGCGGA ACACATGTAA GCGACGGATG TGGCAAAAGT GACGTTTTTG
 181 GTGTGCGCCG GTGTACACAG GAAGTGACAA TTTTCGCGCG GTTTTAGGCG GATGTTGTAG
 241 TAAATTTGGG CGTAACCGAG TAAGATTTGG CCATTTTCGC GGGAAAAC TG AATAAGAGGA
 301 AGTGAAATCT GAATAATTTT GTGTTACTCA TAGCGCGTAA TATTTGTCTA GGGCCGCGGG
 361 GACTTTGACC GTTTACGTGG AGACTCGCCC AGGTGTTTTT CTCAGGTGTT TTCCGCGTTC
 421 CGGGTCAAAG TTGGCGTTTT ATTATTATAG TCAGCTGACG TG TAGTGTAT TTATAACCCG
 481 TGAGTTCCTC AAGAGGCCAC TCTTGAGTGC CAGCGAGTAG AGTTTTCTCC TCCGAGCCGC
 541 TCCGACACCG GGA CTGAAAA TGAGACATGA GGTACTGGCT GATAATCTTC CACCTCCTAG
 601 CCATTTTGAA CCACCTACCC TTCACGAACT GTATGATTTA GACGTGACGG CCCCCGAAGA
 661 TCCAACGAG GAGGCGGTTT CGCAGATTTT TCCC GACTCT GTAATGTTGG CGGTGCAGGA
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 841 TCCACCCAGT GACGACGAGG ATGAAGAGGG TGAGGAGTTT GTGTTAGATT ATGTGGAGCA
 901 CCCC GGGCAG GGTTCAGGT CTTGTCAAT TCACCGGAGG AATACGGGGG ACCCAGATAT
 961 TATGTGTTTC CTTTGCTATA TGAGGACCTG TGGCATGTTT GTCTACAGTA AGTGA AAATT
 1021 ATGGGCAGTG GGTGATAGAG TGGTGGGTTT GGTGTGGTAA TTTTTTTTTT AATTTTTTACA
 1081 GTTTTGTGGT TTAAAGAATT TTGTATTGTG ATTTTTTTTAA AAGGTCCTGT GTCTGAACCT
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 1321 CCCATTAAAC CAGTTGCCGT GAGAGTTGGT GGGCGTCGCC AGGCTGTGGA ATGTATCGAG
 1381 GACTTGCTTA ACGAGCCTGG GCAACCTTTG GACTTGAGCT GTAAACGCCC GAGGCCATAA
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kd1

FIGURE 22
 (SHEET 1)

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9901 TCTATCGCTG CGGCGGCGGC GGAGTTTGGC CGTAGGTGGC GCCCTCTTCC TCCCATGCGT
9961 GTGACCCCGA AGCCCTCAT CGGCTGAAGC AGGGCTAGGT CGGCGACAAC GCGCTCGGCT
10021 AATATGGCCT GCTGCACCTG CGTGAGGGTA GACTGGAAGT CATCCATGTC CACAAAGCGG
10081 TGGTATGCGC CCGTGTTGAT GGTGTAAGTG CAGTTGGCCA TAACGGACCA GTTAAACGGT
10141 TGGTAGCCCG GCTGCGAGAG CTCGGTGTAC CTGAGACGCG AGTAAGCCCT CGAGTCAAA
10201 ACGTAGTCGT TGCAAGTCCG CACCAGGTAC TGGTATCCCA CCAAAAAGTG CGGCGGCGGC
10261 TGGCGGTAGA GGGGCCAGCG TAGGGTGGCC GGGGCTCCGG GGGCGAGATC TTCCAACATA
10321 AGGCGATGAT ATCCGTAGAT GTACCTGGAC ATCCAGGTGA TGCCGGCGGC GGTGGTGGAG
10381 GCGCGCGGAA AGTCGCGGAC GCGGTTCCAG ATGTTGCGCA GCGGCAAAAA GTGCTCCATG
10441 GTCGGGACGC TCTGGCCGGT CAGGCGCGCG CAATCGTTGA CGCTCTAGCG TGCAAAAGGA
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10561 GGACGACCGG GGTTCGAGCC CCGTATCCGG CCGTCCGCGG TGATCCATGC GGTTACCGCC
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12481 GCCGGCCTGG TCTACGACGC GCTGCTTTCAG CGCGTGGCTC GTTACAACAG CCGCAACGTC
12541 CAGACCAACC TGGACCGGCT GGTGGGGGAT GTGCGCGAGG CCGTGGCGCA GCGTGAGCGC
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12721	ATGGTGACTG	AGACACCGCA	AAGTGAGGTG	TACCAGTCTG	GGCCAGACTA	TTTTTTCCAG
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12841	CTGTGGGGGG	TGCGGGCTCC	CACAGGCGAC	CGCGCGACCG	TGTCTAGCTT	GCTGACGCCC
12901	AACTCGCGCC	TGTTGCTGCT	GCTAATAGCG	CCCTTCACGG	ACAGTGGCAG	CGTGTCCCGG
12961	GACACATACC	TAGGTCACCT	GCTGACACTG	TACCGCGAGG	CCATAGGTCA	GGCGCATGTG
13021	GACGAGCATA	CTTTCCAGGA	GATTACAAGT	GTCAGCCGCG	CGCTGGGGCA	GGAGGACACG
13081	GGCAGCCTGG	AGGCAACCCCT	AAACTACCTG	CTGACCAACC	GGCGGCAGAA	GATCCCCTCG
13141	TTGCACAGTT	TAAACAGCGA	GGAGGAGCGC	ATTTTTCGCT	ACGTGCAGCA	GAGCGTGAGC
13201	CTTAACCTGA	TGCGCGACGG	GGTAACGCCC	AGCGTGGCGC	TGGACATGAC	CGCGCGCAAC
13261	ATGGAACCGG	GCATGTATGC	CTCAAACCGG	CCGTTTATCA	ACCGCCTAAT	GGACTACTTG
13321	CATCGCGCGG	CCGCCGTGAA	CCCCGAGTAT	TTCACCAATG	CCATCTTGAA	CCCGCACTGG
13381	CTACCGCCCC	CTGGTTTCTA	CACCGGGGGA	TTCGAGGTGC	CCGAGGGTAA	CGATGGATTG
13441	CTCTGGGACG	ACATAGACGA	CAGCGTGTTC	TCCCCGCAAC	CGCAGACCTT	GCTAGAGTTG
13501	CAACAGCGCG	AGCAGGCAGA	GGCGGCGCTG	CGAAAGGAAA	GCTTCCGCAG	GCCAAGCAGC
13561	TTGTCCGATC	TAGGCGCTGC	GGCCCCGCGG	TCAGATGCTA	GTAGCCCAT	TCCAAGCTTG
13621	ATAGGGTCTC	TTACCAGCAC	TCGCACCACC	CGCCCGCGCC	TGCTGGGCGA	GGAGGAGTAC
13681	CTAAACAAC	CGCTGCTGCA	GCCGCAGCGC	GAAAAAAACC	TGCCTCCGGC	ATTTCCCAAC
13741	AACGGGATAG	AGAGCCTAGT	GGACAAGATG	AGTAGATGGA	AGACGTACGC	GCAGGAGCAC
13801	AGGGACGTGC	CAGGCCCGCG	CCCCGCCACC	CGTCGTCAAA	GGCACGACCG	TCAGCGGGGT
13861	CTGGTGTGGG	AGGACGATGA	CTCGGCAGAC	GACAGCAGCG	TCCTGGATTT	GGGAGGGAGT
13921	GGCAACCCGT	TTGCGCACCT	TCGCCCCAGG	CTGGGGAGAA	TGTTTTAAAA	AAAAAAAAGC
13981	ATGATGCAAA	ATAAAAAACT	CACCAAGGCC	ATGGCACCGA	GCGTTGGTTT	TCTGTATTTC
14041	CCCTTAGTAT	GCGGCGCGCG	GCGATGTATG	AGGAAGGTCC	TCCTCCCTCC	TACGAGATG
14101	TGTTGAGCGC	GGCGCCAGTG	GCGGCGCGCG	TGGGTTCTCC	CTTCGATGCT	CCCCTGGACC
14161	CGCCGTTTGT	GCCTCCGCGG	TACCTGCGGC	CTACCGGGGG	GAGAAACAGC	ATCCGTTACT
14221	CTGAGTTGGC	ACCCCTATTTC	GACACCACCC	GTGTGTACCT	GGTGGACAAC	AAGTCAACGG
14281	ATGTGGCATC	CCTGAACCTAC	CAGAACGACC	ACAGCAACTT	TCTGACCACG	GTCAATCAAA
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14641	AGAACGGGGT	TCTGGAAGC	GACATCGGGG	TAAAGTTTGA	CACCCGCAAC	TTGAGACTGG
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14821	TGTTGGGCAT	CCGCAAGCGG	CAACCCTTCC	AGGAGGGCTT	TAGGATCACC	TACGATGATC
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14941	ATGACACCGA	ACAGGGCGGG	GGTGGCGCAG	GCGGCAGCAA	CAGCAGTGGC	AGCGGCGCGG
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15061	CCATTGCGGG	CGACACCTTT	GCCACACGGG	CTGAGGAGAA	GCGCGCTGAG	GCCGAAGCAG
15121	CGGCCGAAGC	TGCCGCCCCC	GCTGCGCAAC	CCGAGGTGCA	GAAGCCTCAG	AAGAAACCGG
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15241	GCACCTTCAC	CCAGTACCGC	AGCTGGTACC	TTGCATACAA	CTACGGCGAC	CCTCAGACCG
15301	GAATCCGCTC	ATGGACCCTG	CTTTGCACTC	CTGACGTAAC	CTGCGGCTCG	GAGCAGGTCT
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15421	GCAACTTTCC	GGTGGTGGGC	GCCGAGCTGT	TGCCCCGTGCA	CTCCAAGAGC	TTCTACAACG
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15901	GGGGCGCGCA	CAAACGCGGC	CGCACTGGGC	GCACCACCGT	CGATGACGCC	ATCGACGCGG
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16141	TGCTTAACCG	CGCACGTCGC	ACCGGCCGAC	GGGCGGCCAT	GCGGGCCGCT	CGAAGGCTGG
16201	CCGCGGGTAT	TGTCACTGTG	CCCCCAGGT	CCAGGCGACG	AGCGGCCGCG	GCAGCAGCCG
16261	CGGCCATTAG	TGCTATGACT	CAGGGTCGCA	GGGGCAACGT	GTATTGGGTG	CGCGACTCGG
16321	TTAGCGGCCT	GCGCGTGCCC	GTGCGCACCC	GCCCCCGCG	CAACTAGATT	GCAAGAAAAA
16381	ACTACTTAGA	CTCGTACTGT	TGTATGTATC	CAGCGGCGGC	GGCGCGCAAC	GAAGCTATGT
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17401	GACGCCGAAC	CACCACTGGA	ACCCGCCGCC	GCCGTCGCCG	TCGCCAGCCC	GTGCTGGCCC
17461	CGATTTCCGT	GCGCAGGGTG	GCTCGGAAG	GAGGCAGGAC	CCTGGTGCTG	CCAACAGCGC
17521	GCTACCACCC	CAGCATCGTT	TAAAAGCCGG	TCTTTGTGGT	TCTTGCAGAT	ATGGCCCTCA
17581	CCTGCCGCCT	CCGTTTCCCG	GTGCCGGGAT	TCCGAGGAAG	AATGCACCGT	AGGAGGGGCA
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19141	ATGAAGCTGC	TACTGCTCTT	GAAATAAAACC	TAGAAGAAGA	GGACGATGAC	AACGAAGACG
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19261	GTATAAATAT	TACAAAAGGAG	GGTATTCAAA	TAGGTGTCTG	AGGTCAAACA	CCTAAATATG
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19441	CATATGCAAA	ACCCACAAAT	GAAAATGGAG	GGCAAGGCAT	TCTTGTAAG	CAACAAAATG
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28321	ACTGGGATTA	CTTGGGCATG	TGGTGGTTCT	CCATAGCGCT	TATGTTTGTA	TGCCTTATTA
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28441	TCATTGTGCT	ACACCCAAAC	AATGATGGAA	TCCATAGATT	GGACGGACTG	AAACACATGT
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28621	TTTCTCCACA	ATCTAAATGG	AATGTCAGTT	TCCTCCTGTT	CCTGTCCATC	CGCACCCACT
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28741	TATCCATATG	ACACGGAAAC	CGGTCTCTCA	ACTGTGCCTT	TTCTTACTCC	TCCCTTTGTA
28801	TCCCCCAATG	GGTTTCAAGA	GAGTCCCCCT	GGGGTACTCT	CTTTGCGCCT	ATCCGAACCT
28861	CTAGTTACCT	CCAATGGCAT	GCTTGCGCTC	AAAAATGGGCA	ACGGCCTCTC	TCTGGACGAG
28921	GCCGGCAACC	TTACCTCCCA	AAATGTAACC	ACTGTGAGCC	CACCTCTCAA	AAAAACCAAG
28981	TCAAACATAA	ACCTGGAAAT	ATCTGCACCC	CTCACAGTTA	CCTCAGAAAG	CCTAACTGTG
29041	GCTGCCGCCG	CACCTCTAAT	GGTCGCGGGC	AACACACTCA	CCATGCAATC	ACAGGCCCCG
29101	CTAACCCTGC	ACGACTCCAA	ACTTAGCATT	GCCACCCCAAG	GACCCCTCAC	AGTGTGAGAA
29161	GGAAAGCTAG	CCCTGCAAAC	ATCAGGCCCC	CTCACCACCA	CCGATAGCAG	TACCCCTTACT
29221	ATCACTGCCT	CACCCCTCT	AACTACTGCC	ACTGGTAGCT	TGGGCATTGA	CTTGAAAGAG
29281	CCCATTATTA	CACAAAATGG	AAAACTAGGA	CTAAAGTACG	GGGCTCCTTT	GCATGTAACA
29341	GACGACCTAA	ACACTTTGAC	CGTAGCAACT	GGTCCAGGTG	TGACTATTAA	TAATACTTCC
29401	TTGCAAACTA	AAGTTACTGG	AGCCTTGGGT	TTTGATTACAC	AAGGCAATAT	GCAACTTAAT
29461	GTAGCAGGAG	GACTAAGGAT	TGATTCTCAA	AACAGACGCC	TTATACTTGA	TGTTAGTTAT
29521	CCGTTTGATG	CTCAAAACCA	ACTAAATCTA	AGACTAGGAC	AGGGCCCTCT	TTTTATAAAC
29581	TCAGCCCACA	ACTTGGATAT	TAACATAAC	AAAGGCCTTT	ACTTGTTTAC	AGCTTCAAAC
29641	AATTCCAAAA	AGCTTGAGGT	TAACCTAAGC	ACTGCCAAGG	GGTTGATGTT	TGACCGCTACA
29701	GCCATAGCCA	TTAATGCAGG	AGATGGGCTT	GAATTTGGTT	CACCTAATGC	ACCAAACACA
29761	AATCCCCTCA	AAACAAAAT	TGGCCATGGC	CTAGAATTTG	ATTCAAACAA	GGCTATGGTT

FIGURE 22
(SHEET 9)

29821	CCTAAACTAG	GAACTGGCCT	TAGTTTTGAC	AGCACAGGTG	CCATTACAGT	AGGAAACAAA
29881	AATAATGATA	AGCTAACTTT	GTGGACCACA	CCAGCTCCAT	CTCCTAACTG	TAGACTAAAT
29941	GCAGAGAAAG	ATGCTAAACT	CACTTTGGTC	TTAACAAAAT	GTGGCAGTCA	AATACTTGCT
30001	ACAGTTTCAG	TTTTGGCTGT	TAAAGGCAGT	TTGGCTCCAA	TATCTGGAAC	AGTTCAAAGT
30061	GCTCATCTTA	TTATAAGATT	TGACGAAAAT	GGAGTGCTAC	TAAACAATTC	CTTCCTGGAC
30121	CCAGAATATT	GGAACTTTAG	AAATGGAGAT	CTTACTGAAG	GCACAGCCTA	TACAAACGCT
30181	GTTGGATTTA	TGCCTAACCT	ATCAGCTTAT	CCAAAATCTC	ACGGTAAAAC	TGCCAAAAGT
30241	AACATTGTCA	GTCAGTTTAA	CTTAAACGGA	GACAAAACCTA	AACCTGTAAC	ACTAACCAAT
30301	ACACTAAACG	GTACACAGGA	AACAGGAGAC	ACAACCTCAA	GTGCATACCT	TATGTCATTT
30361	TCATGGGACT	GTCTCTGGCCA	CAACTACATT	AATGAAATAT	TTGCCACATC	CTCTTACACT
30421	TTTTTCATACA	TTGCCCAAGA	ATAAAGAATC	GTTTGTGTTA	TGTTTCAACG	TGTTTATTTT
30481	TCAATTGCAG	AAAATTTCAA	GTCATTTTTT	ATTCAGTAGT	ATAGCCCCAC	CACCACATAG
30541	CTTATACAGA	TCACCGTACC	TTAATCAAAC	TCACAGAACC	CTAGTATTCA	ACCTGCCACC
30601	TCCCTCCCAA	CACACAGAGT	ACACAGTCCT	TTCTCCCCGG	CTGGCCTTAA	AAAGCATCAT
30661	ATCATGGGTA	ACAGACATAT	TCTTAGGTGT	TATATTCCAC	ACGGTTTCCT	GTGAGCCAA
30721	ACGCTCATCA	GTGATATTAA	TAAACTCCCC	GGGCAGCTCA	CTTAAGTTCA	TGTCGTGTCT
30781	CAGCTGCTGA	GCCACAGGCT	GCTGTCCAAC	TTGCGGTTGC	TTAACGGGCG	GCGAAGGAGA
30841	AGTCCACGCC	TACATGGGGG	TAGAGTCATA	ATCGTGCATC	AGGATAGGGC	GGTGGTGCTG
30901	CAGCAGCGCG	CGAATAAACT	GCTGCCGCGC	CCGCTCCGTC	CTGCAGGAAT	ACAACATGGC
30961	AGTGGTCTCC	TCAGCGATGA	TTCGCACCCG	CCGCAGCATA	AGGCGCCTTG	TCCTCCGGGC
31021	ACAGCAGCGC	ACCCTGATCT	CACTTAAATC	AGCACAGTAA	CTGCAGCACA	GCACCACAAT
31081	ATTGTTTCAA	ATCCACAGT	GCAAGGCGCT	GTATCCAAAG	CTCATGGCGG	GGACCACAGA
31141	ACCCACGTGG	CCATCATACC	ACAAGCGCAG	GTAGATTAAG	TGGCGACCCC	TCATAAACAC
31201	GCTGGACATA	AACATTACCT	CTTTTGGCAT	GTTGTAATTC	ACCACCTCCC	GGTACCATAT
31261	AAACCTCTGA	TTAAACATGG	CGCCATCCAC	CACCATCCTA	AACCAGCTGG	CCAAAACCTG
31321	CCCGCCGGCT	ATACACTGCA	GGGAACCGGG	ACTGGAACAA	TGACAGTGGA	GAGCCCAGGA
31381	CTCGTAACCA	TGGATCATCA	TGCTCGTCAT	GATATCAATG	TTGGCACAACT	ACAGGCACAC
31441	GTGCATACAC	TTCTCTCAGG	TTACAAGCTC	CTCCCCGCTT	AGAACCATAT	CCAGGGGAAC
31501	AACCCATTCC	TGAATCAGCG	TAAATCCCAC	ACTGCAGGGA	AGACCTCGCA	CGTAACTCAC
31561	GTTGTGCATT	GTCAAAGTGT	TACATTCGGG	CAGCAGCGGA	TGATCCTCCA	GTATGGTAGC
31621	GCGGGTTTCT	GTCTCAAAAG	GAGGTAGACG	ATCCCTACTG	TACGGAGTGC	GCCGAGACAA
31681	CCGAGATCGT	GTTGGTCGTA	GTGTCAATGC	AAATGGAACG	CCGGACGTAG	TCATATTTC
31741	TGAAGCAAAA	CCAGGTGCGG	GCGTGACAAA	CAGATCTGCG	TCTCCGGTCT	CGCCGCTTAG
31801	ATCGCTCTGT	GTAGTAGTTG	TAGTATATCC	ACTCTCTCAA	AGCATCCAGG	CGCCCCCTGG
31861	CTTCGGGTTT	TATGTAAACT	CCTTCATGCG	CCGCTGCCCT	GATAACATCC	ACCACCGCAG
31921	AATAAGCCAC	ACCCAGCCAA	CCTACACATT	CGTTCTGCGA	GTCACACACG	GGAGGAGCGG
31981	GAAGAGCTGG	AAGAACCATT	TTTTTTTTTT	TATTTCAAAA	GATTATCCAA	AACCTCAAAA
32041	TGAAGATCTA	TTAAGTGAAC	GCGCTCCCCT	CCGGTGCGCT	GCTCAAACCT	TACAGCCAAA
32101	GAACAGATAA	TGGCATTTGT	AAGATGTTGC	ACAATGGCTT	CCAAAAGGCA	AACGGCCCTC
32161	ACGTCCAAGT	GGACGTAAAG	GCTAAACCCT	TCAGGGTGAA	TCTCCTCTAT	AAACATTCCA
32221	GCACCTTCAA	CCATGCCCAA	ATAATTCTCA	TCTCGCCACC	TTCTCAATAT	ATCTCTAAGC
32281	AAATCCCGAA	TATTAAGTCC	GGCCATTGTA	AAAATCTGCT	CCAGAGCGCC	CTCCACCTTC
32341	AGCCTCAAGC	AGCGAATCAT	GATTGCAAAA	ATTCAAGTTT	CTCACAGACC	TGTATAAGAT
32401	TCAAAAGCGG	AACATTAACA	AAAATACCGC	GATCCCGTAG	GTCCCTTCGC	AGGGCCAGCT
32461	GAACATAATC	GTGCAGGTCT	GCACGGACCA	GCGCGGCCAC	TTCCCCGCCA	GGACCTTTGA
32521	CAAAAGAAC	CACACTGATT	ATGACAGCGA	TACTCGGAGC	TATGCTAACC	AGCGTAGCCC
32581	CGATGTAAGC	TTTGTGTGAT	GGGCGGCGAT	ATAAAATGCA	AGGTGCTGCT	CAAAAAATCA
32641	GGCAAAGCCT	CTCGCAAAAA	AGAAAGCACA	TCGTAGTCAT	GCTCATGCAG	ATAAAGGCAG
32701	GTAAGCTCCG	GAACCACCAC	AGAAAAAGAC	ACCATTTTTT	TCTCAAACAT	GTCTGCGGGT
32761	TTCTGCATAA	ACACAAAATA	AAATAACAAA	AAAACATTTA	AACATTAGAA	GCCTGTCTTA
32821	CAACAGGAAA	AACAACCCTT	ATAAGCATAA	GACGGACTAC	GGCCATGCCG	GCGTGACCGT
32881	AAAAAACTG	GTCACCGTGA	TTAAAAAGCA	CCACCGACAG	CTCCTCGGTC	ATGTCCGGAG
32941	TCATAATGTA	AGACTCGGTA	AACACATCAG	GTTGATTTCAT	CGGTCAGTGC	TAAAAAGCGA
33001	CCGAAATAGC	CCGGGGGAAT	ACATACCCGC	AGGCGTAGAG	ACAACATTAC	AGCCCCCATA
33061	GGAGGTATAA	CAGAAATTAAT	AGGAGAGAAA	AACACATAAA	CACCTGAAAA	ACCCCTCTGC
33121	CTAGGCAAAA	TAGACCCCTC	CCGCTCCAGA	ACAACATACA	GCGCTTCACA	GCGGCAGCCT
33181	AACAGTCAGC					

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33241 CCAGCTCAAT CAGTCACAGT GTAAAAAAGG GCCAAGTGCA GAGCGAGTAT ATATAGGACT
33301 AAAAAATGAC GTAACGGTTA AAGTCCACAA AAAACACCCA GAAAACCGCA CGCGAACCTA
33361 CGCCCAGAAA CGAAAGCCAA AAAACCCACA ACTTCCTCAA ATCGTCACTT CCGTTTTCCC
33421 ACGTTACGTA ACTTCCCAT TTAAGAAAAC TACAATTCCC AACACATACA AGTTACTCCG
33481 CCCTAAAACC TACGTCACCC GCCCCGTTCC CACGCCCCGC GCCACGTCAC AAAGTCCACC
33541 CCCTCATTAT CATATTGGCT TCAATCCAAA ATAAGGTATA TTATTGATGA TG

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 DEFINITION KD3
 ACCESSION KD3
 KEYWORDS .
 SOURCE Unknown.
 ORGANISM Unknown
 Unclassified.
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 AUTHORS Self
 JOURNAL Unpublished.
 FEATURES Location/Qualifiers
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 ORIGIN

1 CATCATCAAT AATATACCTT ATTTTGGATT GAAGCCAATA TGATAATGAG GGGGTGGAGT
 61 TTGTGACGTG GCGCGGGGCG TGGGAACGGG GCGGGTGACG TAGTAGTGTG GCGGAAGTGT
 121 GATGTTGCAA GTGTGGCGGA ACACATGTAA GCGACGGATG TGGCAAAAGT GACGTTTTTTG
 181 GTGTGCGCCG GTGTACACAG GAAGTGACAA TTTTCGCGCG GTTTTAGGCG GATGTTGTAG
 241 TAAATTTGGG CGTAACCGAG TAAGATTTGG CCATTTTCGC GGGAAACTG AATAAGAGGA
 301 AGTGAAATCT GAATAATTTT GTGTTACTCA TAGCGCGTAA TATTTGTCTA GGGCCGCGGG
 361 GACTTTGACC GTTTACGTGG AGACTCGCCC AGGTGTTTTT CTCAGGTGTT TTCCGCGTTC
 421 CGGGTCAAAG TTGGCGTTTT ATTATTATAG TCAGCTGACG TGTAAGTGTAT TTATACCCGG
 481 TGAGTTCCTC AAGAGGCCAC TCTTGAGTGC CAGCGAGTAG AGTTTTCTCC TCCGAGCCGC
 541 TCCGACACCG GGAAGTAAAA TGAGACATGA GGTACTGGCT GATAATCTTC CACCTCCTAG
 601 CCATTTTGAA CCACCTACCC TTCACGAAC GTATGATTTA GACGTGACGG CCCCCGAAGA
 661 TCCCAACGAG GAGGCGGTTT CGCAGATTTT TCCCAGCTCT GTAATGTTGG CGGTGCAGGA
 721 AGGGATTGAC TTAAGTCACTT TTCCGCGCGC GCGCGGTTCT CCGGAGCCGC CTCACCTTTC
 781 CCGGCAGCCC GAGCAGCCGG AGCAGAGAGC CTTGGGTCCG GTTTGCCACG AGGCTGGCTT
 841 TCCACCCAGT GACGACGAGG ATGAAGAGGG TGAGGAGTTT GTGTTAGATT ATGTGGAGCA
 901 CCGCGGGCAC GGTGTCAGGT CTTGTCAATG TCACCGGAGG AATACGGGGG ACCCAGATAT
 961 TATGTGTTTCG CTTTGCTATA TGAGGACCTG TGGCATGTTT GTCTACAGTA AGTGAATAAT
 1021 ATGGGCAGTG GGTGATAGAG TGGTGGGTTT GGTGTGGTAA TTTTTTTTTT AATTTTTTACA
 1081 GTTTTGTGGT TTAAAGAATT TTGTATTGTG ATTTTTTTTAA AAGGTCTGTG GTCTGAACCT
 1141 GAGCCTGAGC CCGAGCCAGA ACCGGAGCCT GCAAGACCTA CCGCCGTCC TAAAATGGCG
 1201 CCTGCTATCC TGAGACGCCC GACATCACCT GTGTCTAGAG AATGCAATAG TAGTACGGAT
 1261 AGCTGTGACT CCGGTCCCTC TAACACACCT CCTGAGATAC ACCCGGTGGT CCCGCTGTGC
 1321 CCCATTAAAC CAGTTGCCGT GAGAGTTGGT GGGCGTCGCC AGGCTGTGGA ATGTATCGAG
 1381 GACTTGCTTA ACGAGCCTGG GCAACCTTTG GACTTGAGCT GTAAACGCCC CAGGCCATAA
 1441 GGTGTAAACC TGTGATTGCG TGTGTGGTTA ACGCCTTTGT TTGCTGAATG AGTTGATGTA
 1501 AGTTTAAATA AGGGTGAGAT AATGTTTAACT TTGCATGGCG TGTTAAATGG GGCGGGGCTT
 1561 AAAGGGTATA TAATGCGCCG TGGGCTAATC TTGGTTACAT CTGACCTCAT GGAGGCTTGG
 1621 GAGTGTGTTG AAGATTTTTT TGCTGTGCGT AACTTGCTGG AACAGAGCTC TAACAGTACC
 1681 TCTTGGTTTT GGAGGTTTCT GTGGGGCTCA TCCCAGGCAA AGTTAGTCTG CAGAATTAAG
 1741 GAGGATTACA AGTGGGAATT TGAAGAGCTT TTGAAATCCT GTGGTGAGCT GTTTGATTCT
 1801 TTGAATCTGG GTCACCAGGC GCTTTTCCAA GAGAAGGTCA TCAAGACTTT GGATTTTTTCC
 1861 ACACCGGGGC GCGCTGCGGC TGCTGTTGCT TTTTGGAGTT TTATAAAGGA TAAATGGAGC
 1921 GAAGAAACCC ATCTGAGCGG GGGGTACCTG CTGGATTTTC TGGCCATGCA TCTGTGGAGA
 1981 GCGGTTGTGA GACACAAGAA TCGCCTGCTA CTGTTGTCTT CCGTCCGCCC GGCGATAATA
 2041 CCGACGGAGG AGCAGCAGCA GCAGCAGGAG GAAGCCAGGC GCGGCGGCGA GGAGCAGAGC
 2101 CCATGGAACC CGAGAGCCGG CCTGGACCCT CGGGAATGAA TGTTGTACAG GTGGCTGAAC
 2161 TGTATCCAGA ACTGAGACGC ATTTTGACAA TTACAGAGGA TGGGCAGGGG CTAAAGGGGG
 2221 TAAAGAGGGA GCGGGGGGCT TGTGAGGCTA CAGAGGAGGC TAGGAATCTA GCTTTTAGCT
 2281 TAATGACCAG ACACCGTCCT GAGTGTATTA CTTTCAACA GATCAAGGAT AATTGCGCTA
 2341 ATGAGCTTGA TCTGCTGGCG CAGAAGTATT CCATAGAGCA GCTGACCACT TACTGGCTGC
 2401 AGCCAGGGGA TGATTTTGAG GAGGCTATTA GGGTATATGC AAAGGTGGCA CTTAGGCCAG

FIGURE 23
 (SHEET 1)

2461	ATTGCAAGTA	CAAGATCAGC	AAACTTGTA	ATATCAGGAA	TTGTTGCTAC	ATTTCTGGGA
2521	ACGGGGCCGA	GGTGGAGATA	GATACGGAGG	ATAGGGTGCC	CTTTAGATGT	AGCATGATAA
2581	ATATGTGGCC	GGGGGTGCTT	GGCATGGACG	GGGTGGTTAT	TATGAATGTA	AGGTTTACTG
2641	GCCCCAATTT	TAGCGGTACG	GTTTTCCCTG	CCAATACCAA	CCTTATCCTA	CACGGTGTA
2701	GCTTCTATGG	GTTTAACAAT	ACCTGTGTGG	AAGCCTGGAC	CGATGTAAGG	GTTCCGGGCT
2761	GTGCCTTTTA	CTGCTGCTGG	AAGGGGGTGG	TGTGTCGCCC	CAAAAGCAGG	GCTTCAATTA
2821	AGAAATGCCT	CTTTGAAAGG	TGTACCTTGG	GTATCCTGTC	TGAGGGTAAC	TCCAGGGTGC
2881	GCCACAATGT	GGCCTCCGAC	TGTGGTTGCT	TCATGCTAGT	GAAAAGCGTG	GCTGTGATTA
2941	AGCATAACAT	GGTATGTGGC	AACTGCGAGG	ACAGGGCCTC	TCAGATGCTG	ACCTGCTCGG
3001	ACGGCAACTG	TCACCTGCTG	AAGACCATTG	ACGTAGCCAG	CCACTCTCGC	AAGGCCTGGC
3061	CAGTGTTTGA	GCATAACATA	CTGACCCGCT	GTTCCCTGCA	TTTGGGTAAC	AGGAGGGGGG
3121	TGTTCCCTACC	TTACCAATGC	AATTTGAGTC	ACACTAAGAT	ATTGCTTGAG	CCCAGAGCA
3181	TGTCCAAGGT	GAACCTGAAC	GGGGTGTTTG	ACATGACCAT	GAAGATCTGG	AAGGTGCTGA
3241	GGTACGATGA	GACCCGCACC	AGTGCAGAC	CCTGCGAGTG	TGGCGGTAAA	CATATTAGGA
3301	ACCAGCCTGT	GATGCTGGAT	GTGACCGAGG	AGCTGAGGCC	CGATCACTTG	GTGCTGGCCT
3361	GCACCCGCGC	TGAGTTTGGC	TCTAGCGATG	AAGATACAGA	TTGAGGTACT	GAAATGTGTG
3421	GGCGTGGCTT	AAGGGTGGGA	AAGAATATAT	AAGGTGGGGG	TCTTATGTAG	TTTTGTATCT
3481	GTTTTGCAGC	AGCCGCCGCC	GCCATGAGCA	CCAACTCGTT	TGATGGAAGC	ATTGTGAGCT
3541	CATATTTGAC	AACGCGCATG	CCCCCATGGG	CCGGGGTGCG	TCAGAAATGTG	ATGGGCTCCA
3601	GCATTGATGG	TCGCCCCGTC	CTGCCCCGAA	ACTCTACTAC	CTTGACCTAC	GAGACCGTGT
3661	CTGGAACGCC	GTTGGAGACT	GCAGCCTCCG	CCGCCGCTTC	AGCCGCTGCA	GCCACCGCCC
3721	GCGGGATTGT	GACTGACTTT	GCTTTCTCTG	GCCCCGCTTG	AAGCAGTGCA	GCTTCCCGTT
3781	CATCCGCCCG	CGATGACAAG	TTGACGGCTC	TTTTGGCACA	ATTGGATTCT	TTGACCCGGG
3841	AACTTAATGT	CGTTTCTCAG	CAGCTGTTGG	ATCTGCGCCA	GCAGGTTTCT	GCCCTGAAGG
3901	CTTCTCCCC	TCCCAATGCG	GTTTAAAACA	TAAATAAAAA	ACCAGACTCT	GTTTGGATT
3961	GGATCAAGCA	AGTGTCTTGC	TGTCTTTATT	TAGGGGTTTT	GCGCGCGCGG	TAGGCCCGGG
4021	ACCAGCGGTC	TCGGTCGTTG	AGGGTCCTGT	GTATTTTTTC	CAGGACGTGG	TAAAGGTGAC
4081	TCTGGATGTT	CAGATACATG	GGCATAAGCC	CGTCTCTGGG	GTGGAGGTAG	CACCACTGCA
4141	GAGCTTCATG	CTGCGGGGTG	GTGTTGTAGA	TGATCCAGTC	GTAGCAGGAG	CGCTGGGCGT
4201	GGTGCCTAAA	AATGTCTTTC	AGTAGCAAGC	TGATTGCCAG	GGGCAGGCCC	TTGGTGTAAG
4261	TGTTTACAAA	GCGGTAAAGC	TGGGATGGGT	GCATACGTGG	GGATATGAGA	TGCATCTTGG
4321	ACTGTATTTT	TAGGTTGGCT	ATGTTCCCACT	CCATATCCCT	CCGGGGATTG	ATGTTGTGCA
4381	GAACCACGAG	CACAGTGTAT	CCGGTGCACT	TGGGAAATTT	GTCAATGTAGC	TTAGAAGGAA
4441	ATGCGTGGAA	GAACCTGGAG	ACGCCCTTGT	GACCTCCAAG	ATTTTCCATG	CATTTCGTCCA
4501	TAATGATGGC	AATGGGCCCC	CGGGCGGCGG	CCTGGGCGAA	GATATTTCTG	GGATCACTAA
4561	CGTCATAGTT	GTGTTCCAGG	ATGAGATCGT	CATAGGCCAT	TTTTACAAAG	CGCGGGCGGA
4621	GGGTGCCAGA	CTGCGGTATA	ATGTTTCCAT	CCGGCCCAGG	GGCGTAGTTA	CCCTCACAGA
4681	TTTGCATTTT	CCACGCTTTG	AGTTTCAGATG	GGGGGATCAT	GTCTACCTGC	GGGGCGATGA
4741	AGAAAACGGT	TTCCGGGGTA	GGGGAGATCA	GCTGGGAAGA	AAGCAGGTTT	CTGAGCAGCT
4801	GCGACTTACC	GCAGCCGGTG	GGCCCCGTAA	TCACACCTAT	TACCGGGTGC	AACTGGTAGT
4861	TAAGAGAGCT	GCAGCTGCCG	TCATCCCTGA	GCAGGGGGGC	CACTTCGTTA	AGCATGTCCC
4921	TGACTCGCAT	GTTTTCCCTG	ACCAAATCCG	CCAGAAGGCG	CTCGCCGCCC	AGCGATAGCA
4981	GTTCTTGCAA	GGAAGCAAAG	TTTTTCAACG	GTTTGAGACC	GTCCGCCGTA	GGCATGCTTT
5041	TGAGCGTTTG	ACCAAGCAGT	TCCAGGCGGT	CCCACAGCTC	GGTCACCTGC	TCTACGGCAT
5101	CTCGATCCAG	CATATCTCCT	CGTTTTCGCG	GTTGGGGCGG	CTTTTCGCTG	ACGGCAGTAG
5161	TCGGTGCTCG	TCCAGACGGG	CCAGGGTCAT	GTCTTTCCAC	GGGCGCAGGG	TCCTCGTCAG
5221	CGTAGTCTGG	GTCACGGTGA	AGGGGTGCGC	TCCGGGCTGC	GCGCTGGCCA	GGGTGCGCTT
5281	GAGGCTGGTC	CTGCTGGTGC	TGAAGCGCTG	CCGGTCTTCG	CCCTGCGCGT	CGGCCAGGTA
5341	GCATTTGACC	ATGGTGTATC	AGTCCAGCCC	CTCCGCGGCG	TGGCCCTTGG	CGCGCAGCTT
5401	GCCCTTGGAG	GAGCGCCCG	ACGAGGGGCA	GTGCAGACTT	TTGAGGGCGT	AGAGCTTGGG
5461	CGCGAGAAAT	ACCGATTCCG	GGGAGTAGGC	ATCCGCGCCG	CAGGCCCCGC	AGACGGTCTC
5521	GCATTCCACG	AGCCAGGTGA	GCTCTGGCCG	TTCGGGGTCA	AAAACCAGGT	TTCCCCCATG
5581	CTTTTTGATG	CGTTTCTTAC	CTCTGGTTTC	CATGAGCCGG	TGTCCACGCT	CGGTGACGAA
5641	AAGGCTGTCC	GTGTCCCCGT	ATACAGACTT	GAGAGGCCTG	TCCTCGAGCG	GTGTTCCGCG
5701	GTCCTCCTCG	TATAGAAACT	CGGACCACTC	TGAGACAAAG	GCTCGCGTCC	AGGCCAGCAC
5761	GAAGGAGGCT	AAGTGGGAGG	GGTAGCGGTC	GTTGTCCACT	AGGGGGTCCA	CTCGCTCCAG
5821	GGTGTGAAGA	CACATGTGCG	CCTCTTCGGC	ATCAAGGAAG	GTGATTGGTT	TGTAGGTGTA

FIGURE 23
(SHEET 2)

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5881 GGCCACGTGA CCGGGTGTTC CTGAAGGGGG GCTATAAAAG GGGGTGGGGG CGCGTTCGTC
5941 CTCACCTCTT TCCGCATCGC TGTCTGCGAG GGCCAGCTGT TGGGGTGAGT ACTCCCTCTG
6001 AAAAGCGGGG ATGACTTCTG CGCTAAGATT GTCAGTTTCC AAAAACGAGG AGGATTTGAT
6061 ATTCACCTGG CCGCGGTGA TGCCTTTGAG GGTGGCCGCA TCCATCTGGT CAGAAAAAGAC
6121 AATCTTTTTT TTGTCAAGCT TGGTGGCAAA CGACCCGTAG AGGGCGTTGG ACAGCAACTT
6181 GGCGATGGAG CGCAGGGTTT GGTTTTTGTG GCGATCGGCG CGCTCCTTGG CCGCGATGTT
6241 TAGCTGCACG TATTCGCGCG CAACGCACCG CCATTTCGGA AAGACGGTGG TGCCTCTGTC
6301 GGGCACCAGG TGCACGCGCC AACC CGGTT GTGCAGGGTG ACAAGGTCAA CGCTGGTGGC
6361 TACCTCTCCG CGTAGGCGCT CGTTGGTCCA GCAGAGGCGG CCGCCCTTGC GCGAGCAGAA
6421 TGGCGGTAGG GGGTCTAGCT GCGTCTCGTC CGGGGGGTCT GCGTCCACGG TAAAGACCCC
6481 GGGCAGCAGG CGCGCGTCGA AGTAGTCTAT CTTGCATCCT TGCAAGTCTA GCGCCTGCTG
6541 CCATGCGCGG GCGGCAAGCG CGCGCTCGTA TGGGTTGAGT GGGGGACCCC ATGGCATGGG
6601 GTGGGTGAGC GCGGAGGCGT ACATGCCGTA AATGTCGTAA ACGTAGAGGG GCTCTCTGAG
6661 TATTTCCAAGA TATGTAGGGT AGCATCTTCC ACCGCGGATG CTGGCGCGCA CGTAATCGTA
6721 TAGTTCGTGC GAGGGAGCGA GGAGGTCGGG ACCGAGGTTG CTACGGGCGG GCTGCTCTGC
6781 TCGGAAGACT ATCTGCCTGA AGATGGCATG TGAGTTGGAT GATATGGTTG GACGCTGGAA
6841 GACGTTGAAG CTGGCGTCTG TGAGACCTAC CGCGTCACGC ACGAAGGAGG CGTAGGAGTC
6901 GCGCAGCTTG TTGACCAGCT CGGCGGTGAC CTGCACGTCT AGGGCGCAGT AGTCCAGGGT
6961 TTCCTTGATG ATGTCATACT TATCCTGTCC CTTTTTTTTT CACAGCTCGC GGTGAGGAC
7021 AAACCTCTTC CGGTCTTTCC AGTACTCTTG GATCGGAAAC CCGTCGGCCT CCGAACGGTA
7081 AGAGCCTAGC ATGTAGAACT GGTTGACGGC CTGGTAGGCG CAGCATCCCT TTTCTACGGG
7141 TAGCGCGTAT GCCTGCGCGG CCTTCCGGAG CGAGGTGTGG GTGAGCGCAA AGGTGTCCCT
7201 GACCATGACT TTGAGGTACT GGTATTTGAA GTCAGTGTCT TCGCATCCCG GCTCTCTCCA
7261 GAGCAAAAAG TCCGTGCGCT TTTTGGAACG CGGATTTGGC AGGGCGAAGG TGACATCGTT
7321 GAAGAGTATC TTTCCGCGCG GAGGCATAAA GTTGCGTGTG ATGCGGAAGG GTCCCGGCAC
7381 CTCGGAACGG TTGTTAATTA CCTGGGCGGC GAGCACGATC TCGTCAAAGC CGTTGATGTT
7441 GTGGCCCACA ATGTAAAGTT CCAAGAAGCG CGGGATGCCC TTGATGGAAG GCAATTTTTT
7501 AAGTTCCTCG TAGGTGAGCT CTTCAGGGGA GCTGAGCCCG TGCTCTGAAA GGGCCCAGTC
7561 TGCAAGATGA GGGTTGGAAG CGACGAATGA GCTCCACAGG TCACGGGCCA TTAGCATTTG
7621 CAGGTGGTTC CGAAAGGTCC TAAACTGGCG ACCTATGGCC ATTTTTTCTG GGGTGATGCA
7681 GTAGAAGGTA AGCGGGTCTT GTTCCCAGCG GTCCCATCCA AGGTTTCGCG CTAGGTCTCG
7741 CGCGGCAGTC ACTAGAGCTT CATCTCCGCC GAACCTCATG ACCAGCATGA AGGGCACGAG
7801 CTGCTTCCCA AAGGCCCCCA TCCAAGTATA GGTCTCTACA TCGTAGGTGA CAAAGAGACG
7861 CTCGGTGCGA GGATGCGAGC CGATCGGGAA GAACTGGATC TCCCGCCACC AATTGGAGGA
7921 GTGGCTATTG ATGTGGTGAA AGTAGAAGTC CCTGCGACGG GCCGAACACT CGTGCTGGCT
7981 TTTGTAAAAA CGTGCGCAGT ACTGGCAGCG GTGCACGGGC TGTACATCCT GCACGAGGTT
8041 GACCTGACGA CCGCGCACAA GGAAGCAGAG TGGAATTTG AGCCCCTCGC CTGGCGGGTT
8101 TGGCTGGTGG TCTTCTACTT CGGCTGCTTG TCCTTGACCG TCTGGCTGCT CGAGGGGAGT
8161 TACGGTGGAT CGGACCACCA CGCCGCGCGA GCCCAAAGTC CAGATGTCCG CGCGCGGCGG
8221 TCGGAGCTTG ATGACAACAT CGCGCAGATG GGAGCTGTCC ATGGTCTGGA GCTCCGCGG
8281 CGTCAGGTCA GCGGGGAGCT CCTGCAAGTT TACCTCGCAT AGACGGGTCA GGGCGCGGGC
8341 TAGATCCAGG TGATACCTAA TTTCCAGGGG CTGGTTGGTG GCGGCGTCGA TGGCTTGCAA
8401 GAGGCCGCAT CCCC GCGCG CGACTACGGT ACCGCGCGGC GGGCGGTGGG CCGCGGGGGT
8461 GTCCTTGATG GATGCATCTA AAAGCGGTGA CGCGGGCGAG CCCCCGAGG TAGGGGGGGC
8521 TCCGACCCCG CCGGAGAGG GGGCAGGGGC ACGTGCGCGC CGCGCGCGGG CAGGAGCTGG
8581 TGCTGCGCGC GTAGGTTGCT GGCGAACGCG ACGACGCGGC GGTTGATCTC CTGAATCTGG
8641 CGCCTCTGCG TGAAGACGAC GGGCCCGGTG AGCTTGAGCC TGAAAGAGAG TTCGACAGAA
8701 TCAATTTCCG TGTCGTTGAC GGCGGCCTGG CGCAAATCT CCTGCACGTC TCCTGAGTTG
8761 TCTTGATAGG CGATCTCGGC CATGAACTGC TCGATCTCTT CCTCCTGGAG ATCTCCGCGT
8821 CCGGCTCGCT CCACGGTGCG GCGGAGTCTG TTGGAAATGC GGGCCATGAG CTGCGAGAAG
8881 GCGTTGAGGC CTCCCTCGTT CCAGACGCGG CTGTAGACCA CGCCCCCTTC GGCATCGCGG
8941 GCGCGCATGA CCACCTGCGC GAGATTGAGC TCCACGTGCC GGGCGAAGAC GGCGTAGTTT
9001 CGCAGGCGCT GAAAGAGGTA GTTGAGGGTG GTGGCGGTGT GTTCTGCCAC GAAGAAGTAC
9061 ATAACCCAGC GTCGCAACGT GGATTCGTTG ATATCCCCCA AGGCCTCAAG GCGCTCCATG
9121 GCCTCTGAGA AGTCCACGGC GAAGTTGAAA AACTGGGAGT TGCGCGCCGA CACGGTTAAC
9181 TCCTCTCCA GAAGACGGAT GAGCTCGGCG ACAGTGTGCG GCACCTCGCG CTCAAAGGCT
9241 ACAGGGGCCCT CTTCTTCTTC TTCAATCTCC TCTTCCATAA GGGCCTCCCC TTCTTCTTCT

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FIGURE 23
(SHEET 3)

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9301 TCTGGCGGCG GTGGGGGAGG GGGGACACGG CGGCGACGAC GGGCGACCGG GAGGCGGTCTG
9361 ACAAAGCGCT CGATCATCTC CCCGCGGCGA CGGCGCATGG TCTCGGTGAC GGGCGGGCCG
9421 TTCTCGCGGG GCGCGAGTTG GAAGACGCCG CCCGTCATGT CCCGGTTATG GGTTGGCGGG
9481 GGGCTGCCAT GCGGCAGGGA TACGGCGCTA ACGATGCATC TCAACAATTG TTGTGTAGGT
9541 ACTCCGCCGC CGAGGGACCT GAGCGAGTCC GCATCGACCG GATCGGAAAA CCTCTCGAGA
9601 AAGGCGTCTA ACCAGTCACA GTCGCAAGGT AGGCTGAGCA CCGTGGCGGG CGGCAGCGGG
9661 CGGCGGTCTG GGTGTGTTCT GCGCGAGGTG CTGCTGATGA TGTAATTAAA GTAGGCGGTCT
9721 TTGAGACGGC GGATGGTCGA CAGAAGCACC ATGTCCTTGG GTCCGGCCTG CTGAATGCGC
9781 AGGCGGTCTG CCATGCCCCA GGCTTCGTTT TGACATCGGC GCAGGTCTTT GTAGTAGTCT
9841 TGCATGAGCC TTTCTACCGG CACTTCTTCT TCTCCTTCCT CTTGTCTGTC ATCTCTTGCA
9901 TCTATCGCTG CGGCGGCGGC GGAGTTTGGC CGTAGGTGGC GCCCTCTTCC TCCCATGCGT
9961 GTGACCCCGA AGCCCCCTCAT CGGTGAAGC AGGGCTAGGT CGGCGACAAC GCCTCTGGCT
10021 AATATGGCCT GCTGCACCTG CGTGAGGGTA GACTGGAAGT CATCCATGTC CACAAAGCGG
10081 TGGTATGCGC CCGTGTTGAT GGTGTAAGTG CAGTTGGCCA TAACGGACCA GTTAACGGTC
10141 TGGTGACCCG GCTGCGAGAG CTCGGTGTAC CTGAGACGCG AGTAAGCCCT CGAGTCAAAT
10201 ACGTAGTCGT TGCAAGTCCG CACCAGGTAC TGGTATCCCA CCAAAAAGTG CGGCGGCGGC
10261 TGGCGGTAGA GGGGCCAGCG TAGGGTGGCC GGGGCTCCGG GGGCGAGATC TTCCAACATA
10321 AGGCGATGAT ATCCGTAGAT GTACCTGGAC ATCCAGGTGA TGCCGGCGGC GGTGGTGGAG
10381 GCGCGCGGAA AGTCGCGGAC GCGGTTCCAG ATGTTGCGCA GCGGCAAAAA GTGCTCCATG
10441 GTCGGGACGC TCTGGCCGGT CAGGCGCGCG CAATCGTTGA CGCTCTAGCG TGCAAAAAGGA
10501 GAGCCTGTAA GCGGGCACTC TTCCGTGGTC TGGTGGATAA ATTGCAAGG GTATCATGGC
10561 GGACGACCGG GGTTCGAGCC CCGTATCCGG CCGTCCGCGG TGATCCATGC GGTACCGCC
10621 CGCGTGTCGA ACCCAGGTGT GCGACGTGAG ACAACGGGGG AGTGCTCCTT TTGGCTTCCT
10681 TCCAGGCGCG GCGGCTGCTG CGCTAGCTTT TTTGGCCACT GGCCGCGCGC AGCGTAAGCG
10741 GTTAGGCTGG AAAGCGAAAG CATTAAAGTG CTCGCTCCCT GTAGCCGGAG GGTATTTTTC
10801 CAAGGGTTGA GTCGCGGGAC CCCCAGTTTC AGTCTCGGAC CGGCCGGACT GCGGCGAAGC
10861 GGGGTTTGCC TCCCCGTCAT GCAAGACCCC GCTTGCAAAT TCCTCCGGAA ACAGGGACGA
10921 GCCCTTTTTT TGCTTTTCCC AGATGCATCC GGTGCTGCGG CAGATGCGCC CCCCTCCTCA
10981 GCAGCGGCAA GAGCAAGAGC AGCGGCAGAC ATGCAGGGCA CCCTCCCCTC CTCCTACCGC
11041 GTCAGGAGGG GCGACATCCG CGGTTGACGC GGCAGCAGAT GGTGATTACG AACCCCCGCG
11101 GCGCCGGGCC CGGCACTACC TGGACTTGGA GGAGGGCGAG GGCCTGGCGG GCGTAGGAGC
11161 GCCCTCTCCT GAGCGGTACC CAAGGTTGCA GCTGAAGCGT GATACGCGTG AGGCTAGCGT
11221 GCCGCGGCGA AACCTGTTTC GCGACCGCGA GGGAGAGGAG CCCGAGGAGA TGCGGGATCG
11281 AAAGTTCCAC GCAGGGCGCG AGCTGCGGCA TGGCCTGAAT CGCGAGCGGT TGCTGCGCGA
11341 GGAGGACTTT GAGCCCGACG CGCGAACCGG GATTAGTCCC GCGCGCGCAC ACGTGGCGGC
11401 CGCCGACCTG GTAACCGCAT ACGAGCAGAC GGTGAACCAG GAGATTAACT TTCAAAAAAG
11461 CTTTAACAAC CACGTGCGTA CGCTTGTTGGC GCGCGAGGAG GTGGCTATAG GACTGATGCA
11521 TCTGTGGGAC TTTGTAAGCG CGCTGGAGCA AAACCCAAAT AGCAAGCCGC TCATGGCGCA
11581 GCTGTTCTTT ATAGTGACGC ACAGCAGGGA CAACGAGGCA TTCAGGGATG CGCTGCTAAA
11641 CATAGTAGAG CCCGAGGGCC GCTGGCTGCT CGATTGATA AACATCCTGC AGACATAGT
11701 GGTGCAGGAG CGCAGCTTGA GCCTGGCTGA CAAGGTGGCC GCCATCAACT ATTCCATGCT
11761 TAGCCTGGGC AAGTTTACG CCCGCAAGAT ATACCATAAC CCTTACGTTT CCATAGACAA
11821 GGAGGTAAAG ATCGAGGGGT TCTACATGCG CATGGCGCTG AAGGTGCTTA CCTTGAGCGA
11881 CGACCTGGGC GTTTATCGCA ACGAGCGCAT CCACAAGGCC GTGAGCGTGA GCCGGCGGCG
11941 CGAGCTCAGC GACCGCGAGC TGATGCACAG CCTGCAAAGG GCCCTGGCTG GCACGGGCGC
12001 CGGCGATAGA GAGGCCGAGT CCTACTTTGA CGCGGGCGCT GACCTGCGCT GGGCCCCAAG
12061 CCGACGCGCC CTGGAGGCGA CTGGGGCCCG ACCTGGGCTG GCGGTGGCAC CCGCGCGCGC
12121 TGGCAACGTC GCGGCGGTGG AGGAATATGA CGAGGACGAT GAGTACGAGC CAGAGGACGG
12181 CGAGTACTAA GCGGTGATGT TTCTGATCAG ATGATGCAAG ACGCAACGGA CCCGGCGGTC
12241 CGGGCGGCGC TGCAGAGCCA GCCGTCCGCG CTTAACTCCA CGGACGACTG GCGCCAGGTC
12301 ATGGACCGCA TCATGTCGCT GACTGCGCGC AATCCTGACG CGTTCCGGCA GCAGCCGCGC
12361 GCCAACCGGC TCTCCGCAAT TCTGGAAGCG GTGGTCCCGG CGCGCGCAAA CCCACGCGC
12421 GAGAAGGTGC TGGCGATCGT AAACGCGCTG GCCGAAAACA GGGCCATCCG GCCCGACGAG
12481 GCCGGCCTGG TCTACGACGC GCTGCTTCAG CGCGTGGCTC GTTACAACAG CGGCAACGTC
12541 CAGACCAACC TGGACCGGCT GGTGGGGGAT GTGCGCGAGG CCGTGGCGCA GCGTGAGCGC
12601 GCGCAGCAGC AGGGCAACCT GGGCTCCATG GTTGCACTAA ACGCCTTCCT GAGTACACAG
12661 CCCGCCAACG TGCCGCGGGG ACAGGAGGAC TACACCAACT TTGTGAGCGC ACTGCGGCTA

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FIGURE 23
(SHEET 4)

12721	ATGGTGA	CTG	AGACAC	CGCA	AAGTGA	GAGGTG	TACCAG	TCTG	GGCCAG	ACTA	TTTTTT	CCAG	
12781	ACCAGT	AGAC	AAGGCC	TGCA	GACCGT	AAAC	CTGAGC	CAGG	CTTTCA	AAAA	CTTGCA	AGGGG	
12841	CTGTGG	GGGG	TGCGGG	CTCC	CACAGG	CGAC	CGCGCG	ACCG	TGTCTA	GCCTT	GCTGAC	CGCCC	
12901	AAC	TCGCGC	TGTTG	CTGCT	GCTAAT	AGCG	CCCTTC	ACGG	ACAGT	TGGCAG	CGTGT	CCCCG	
12961	GACACAT	ACC	TAGGTC	CACTT	GCTGAC	ACTG	TACCGC	GAGG	CCATAG	GTCA	GGCGCA	TGTG	
13021	GACGAG	CATA	CTTTCC	AGGA	GATTACA	AGT	GTCAGC	CCG	CGCTGG	GGGCA	GGAGGA	CACG	
13081	GGCAGC	CTGG	AGGCA	ACCC	AAACTA	CC	CTGACCA	ACC	GGCGGC	CAGAA	GATCCCC	CTCG	
13141	TTGCAC	AGTT	TAAAC	AGCGA	GGAGGA	GC	ATTTT	TGCG	ACGTGC	CAGCA	GAGCGT	GAGC	
13201	CTTAAC	CTGA	TGCGCG	ACGG	GGTAAC	GCCC	AGCGT	TGGCG	TGGAC	ATGAC	CGCGCG	CAAC	
13261	ATGGA	ACCGG	GCATGT	ATGC	CTCAA	ACCGG	CCGTTT	TATCA	ACCGC	CTAAT	GGACTA	CTTG	
13321	CATCGC	CGCG	CCGCCG	TGAA	CCCCGA	GTAT	TTCA	CCAATG	CCATCT	TGAA	CCCGCA	CTGG	
13381	CTACCG	CCCC	CTGGTT	TCTA	CACCGG	GGGA	TTCGAG	GTG	CCGAGG	GTAA	CGATGG	ATTG	
13441	CTCTGG	GACG	ACATAG	ACGA	CAGCGT	GT	TCCCCG	CAAC	CGCAG	ACCC	GCTAG	ATTG	
13501	CAACAG	CGCG	AGCAGG	CAGA	GGCGGC	GTG	CGAAAG	GAAA	GC	TCCG	CAG	GCCAA	GCAGC
13561	TTGTCC	GATC	TAGGCG	CTGC	GGCCCC	CGCG	TCAGAT	GCTA	G	TAGCCC	ATT	TCCAAG	CTTG
13621	ATAGGG	TCTC	TTACC	CAGCAC	TCGCAC	CACC	CGCCCC	CGCG	TGCTGG	GGCGA	GGAGGA	GAGTAC	
13681	CTAAACA	ACT	CGCTGC	TGCA	GCCGC	CAGCGC	GAAAAA	AACC	TGCC	TCCGGC	ATTTCC	CAAC	
13741	AACGGG	ATAG	AGAGCC	TAGT	GGACA	AAGATG	AGTAG	ATGGA	AGACG	TACGC	GCAGGA	GACAC	
13801	AGGGAC	GTGC	CAGGCC	CGCG	CCCCGC	CACC	CGTCGT	CAAA	GGCAC	GACCG	TCAGCG	GGGGT	
13861	CTGGT	GTGGG	AGGAC	GATGA	CTCGG	CAGAC	GACAGC	CAGCG	TCCTGG	ATT	GGGAGG	GAGT	
13921	GGCAAC	CCCGT	TTGCGC	CACCT	TCGCCC	CAGG	CTGGGG	GAGAA	TGTTTT	TAAAA	AAAAAA	AAGC	
13981	ATGATG	CAAA	ATAAAAA	ACT	CACCA	AAGGCC	ATGGC	ACCGA	GC	TTGG	TTT	TC	TTGTATTC
14041	CCCTT	TAGTAT	GCGGCG	CGCG	GCGATG	TATG	AGGAAG	GTCC	TCCTCC	CTC	TACGAG	AGTG	
14101	TGGTGA	GC	GGCGCC	AGTG	GCGGCG	CGCG	TGGGTT	CTCC	CTTCG	ATGCT	CCCC	TGGACC	
14161	CGCCGT	TTGT	GCCTCC	CGCG	TACCTG	CGGC	CTACC	GGGG	GAGAA	ACAGC	ATCCGT	TACT	
14221	CTGAGT	TGGC	ACCCCT	TATTC	GACACC	ACCC	GTGTGT	TACCT	GGTGG	ACAAC	AAGTCA	ACCG	
14281	ATGTGG	CATC	CCTGA	ACTAC	CAGAAC	GACC	ACAGCA	ACTT	TCTGA	CCACG	GTCA	TTCAAA	
14341	ACAATG	ACTA	CAGCCC	GGGG	GAGGCA	AAGCA	CACAG	ACCAT	CAATCT	TGAC	GACCGG	TCGC	
14401	ACTGGG	GGCGG	CGACCT	TGAAA	ACCATC	CTGC	ATACCA	ACAT	GCCAA	ATGTG	AACGAG	TTCA	
14461	TGTTT	TACCAA	TAAGTT	TAAAG	GCGCGG	GTGA	TGGTGT	TCGCG	CTTGCC	TACT	AAGGAC	AATC	
14521	AGGTGG	GAGCT	GAAAT	ACGAG	TGGGT	TGGAGT	TCACG	CTGCC	CGAGGG	CAAC	TACTCC	GAGA	
14581	CCATG	ACCAT	AGACCT	TATG	AACAAC	CGGA	TCGTGG	AGCA	CTACTT	TGAAA	TGGGG	CAGAC	
14641	AGAACG	GGGT	TCTGGA	AAGC	GACATC	GGGG	TAAAGT	TTGA	CACCCG	CAAC	TTCAG	ACTGG	
14701	GGTTT	GACCC	CGTCA	CTGGT	CTTGT	CATGC	CTGGGG	TATA	TACAA	ACGAA	GCCTT	CCATC	
14761	CAGAC	ATCAT	TTTGT	TGCCA	GGATG	CGGGG	TGGACT	TCAC	CCACAG	CCGC	CTGAG	CAACT	
14821	TGTTGG	GAT	CCGCA	AAGCGG	CAACCT	TTCC	AGGAGG	GGCTT	TAGGAT	CACC	TACGAT	GATC	
14881	TGGAGG	GTGG	TAACAT	TCCC	GCACTG	TGG	ATGTGG	ACGC	CTACC	AGGCG	AGCTTG	AAAG	
14941	ATGAC	ACCGA	ACAGGG	CGGG	GGTGG	CGCAG	GCGGC	AGCAA	CAGCAG	TGGC	AGCGGC	CGCG	
15001	AAGAGA	AACTC	CAACGC	CGGCA	GCCGC	CGGCAA	TGCAGC	CGGT	GGAGGA	CATG	AACGAT	CATG	
15061	CCATT	TCGCGG	CGACAC	CTTT	GCCAC	ACGGG	CTGAGG	GAGAA	GCGCG	CTGAG	GCCGA	AAGCAG	
15121	CGGCCG	AAGC	TGCCG	CCCCC	GCTGC	GCAAC	CCGAGG	TCGA	GAAGCC	TCAG	AAGAA	ACCGG	
15181	TGATCA	AACC	CCTGAC	AGAG	GACAGC	AAAGA	AACGCA	GTTA	CAACCT	TAATA	AGCAAT	GACA	
15241	GCACCT	TCAC	CCAGT	ACCGC	AGCTGG	TACC	TTGCAT	ACAA	CTACGG	CGAC	CCTCAG	ACCG	
15301	GAATCC	GCTC	ATGGAC	CCCTG	CTTTG	CACTC	CTGACG	TAAC	CTGCGG	CTCG	GAGCAG	GTCT	
15361	ACTGGT	CTGTT	GCCAG	ACATG	ATGCA	AAGACC	CCGTGA	CCCTT	CCGCT	CCACG	CGCCAG	ATCA	
15421	GCAACT	TTTCC	GGTGGT	TGGG	GCCGAG	CTGT	TGCCCC	GTGA	CTCCA	AAGAGC	TTCTAC	AACG	
15481	ACCAGG	CCCGT	CTACT	CCCCA	CTCAT	CCGCC	AGTTT	TACCTC	TCTGA	CCCCAC	GTGTT	CAATC	
15541	GCTTT	CCCCG	GAACC	AGATT	TTGGC	CGCGC	CGCCAG	CCCC	CACCAT	CACC	ACCGT	CAGTG	
15601	AAAACG	TTCC	TGCTCT	CACA	GATCAC	GGGA	CGCTAC	CGCT	GCCCA	ACAGC	ATCGG	AGGAG	
15661	TCCAGC	GAGT	GACCAT	TACT	GACGCG	AGAC	GCCGC	ACCTG	CCCC	TACGTT	TACAAG	GGCCC	
15721	TGGGC	ATAGT	CTCGCC	CGCG	GTCCT	ATCGA	GCCGCA	CTTT	TTGAG	CAAGC	ATGTCC	ATCC	
15781	TTATAT	CGCC	CAGCA	ATAAC	ACAGG	CTGGG	GCCTG	CGCTT	CCCAAG	CAAG	ATGTTT	TGGCG	
15841	GGGCCA	AAGAA	GCGCT	CCGAC	CAACACC	CAG	TGCGCG	TGCG	CGGGC	ACTAC	CGCGCG	CCCT	
15901	GGGGCG	CGCA	CAAACG	CGGC	CGCACT	TGGG	GCACC	ACCGT	CGATG	ACGCC	ATCGAC	CGCG	
15961	TGGTGG	AGGA	GGCGCG	CAAC	TACACG	CCCA	CGCCG	CCACC	AGTGT	CCACA	GTGGAC	CGCG	
16021	CCATT	CAGAC	CGTGGT	TGCG	GGAGCC	CGGC	GCTATG	CTAA	AATGA	AAGAGA	CGGCGG	AGGC	
16081	GCGTAG	CACG	TCGCC	ACCGC	CGCCG	ACCCG	GCACTG	CCGC	CCAAC	CGCGC	GCGGCG	GGCC	

16141	TGCTTAACCG	CGCACGTGCG	ACCGGCCGAC	GGGCGGCCAT	GCGGGCCGCT	CGAAGGCTGG
16201	CCGCGGGTAT	TGTCACGTGT	CCCCCAGGT	CCAGGCGACG	AGCGGCCGCC	GCAGCAGCCG
16261	CGGCCATTAG	TGCTATGACT	CAGGGTCGCA	GGGGCAACGT	GTATTGGGTG	CGCGACTCGG
16321	TTAGCGGCCT	GCGCGTGCCC	GTGCGCACCC	GCCCCCGCG	CAACTAGATT	GCAAGAAAAA
16381	ACTACTTAGA	CTCGTACTGT	TGTATGTATC	CAGCGGCGGC	GGCGCGCAAC	GAAGCTATGT
16441	CCAAGCGCAA	AATCAAAGAA	GAGATGCTCC	AGGTCATCGC	GCCGGAGATC	TATGGCCCCC
16501	CGAAGAAGGA	AGAGCAGGAT	TACAAGCCCC	GAAAGCTAAA	GCGGGTCAAA	AAGAAAAAGA
16561	AAGATGATGA	TGATGAACTT	GACGACGAGG	TGGAAGTGT	GCACGCTACC	GCGCCAGGC
16621	GACGGGTACA	GTGGAAAGGT	CGACGCGTAA	AACGTGTTTT	GCGACCCGGC	ACCACCGTAG
16681	TCTTTACGCC	CGGTGAGCGC	TCCACCCGCA	CCTACAAGCG	CGTGTATGAT	GAGGTGTACG
16741	GCGACGAGGA	CCTGCTTGAG	CAGGCCAACG	AGCGCCTCGG	GGAGTTTGCC	TACGGAAAGC
16801	GGCATAAGGA	CATGCTGGCG	TTGCCGCTGG	ACGAGGGCAA	CCCAACACCT	AGCCTAAAGC
16861	CCGTAACACT	GCAGCAGGTG	CTGCCCCGCG	TTGCACCGTC	CGAAGAAAAG	CGCGGCCTAA
16921	AGCGCGAGTC	TGGTGACTTG	GCACCCACCG	TGCAGCTGAT	GGTACCCAAG	CGCCACGCGA
16981	TGGAAGATGT	CTTGGAAAAA	ATGACCGTGG	AACCTGGGCT	GGAGCCCGAG	GTCCGCGTGC
17041	GGCCAATCAA	GCAGGTGGCG	CCGGGACTGG	GCGTGCAGAC	CGTGGACGTT	CAGATACCCA
17101	CTACCAGTAG	CACCAGTATT	GCCACCGCCA	CAGAGGGCAT	GGAGACACAA	ACGTCCCCGG
17161	TTGCCTCAGC	GGTGGCGGAT	GCCGCGGTGC	AGGCGGTGCG	TGCGGCCGCG	TCCAAGACCT
17221	CTACGGAGGT	GCAAACGGAC	CCGTGGATGT	TTCGCGTTTC	AGCCCCCGCG	CGCCCCGCGC
17281	GTTTCGAGGAA	GTACGGCGCC	GCCAGCGCGC	TACTGCCCGA	ATATGCCCTA	CATCCTTCCA
17341	TTGCGCCTAC	CCCCGGCTAT	CGTGGCTACA	CCTACCGCCC	CAGAAGACGA	GCAACTACCC
17401	GACGCCGAAC	CACCACTGGA	ACCCGCCGCC	GCCGTCGCCG	TCGCCAGCCC	GTGCTGGCCC
17461	CGATTTCCGT	GCGCAGGGTG	GCTCGCGAAG	GAGGCAGGAC	CCTGGTGTCTG	CCAACAGCGC
17521	GCTACCACCC	CAGCATCGTT	TAAAAGCCGG	TCTTTGTGGT	TCTTGCAGAT	ATGGCCCTCA
17581	CCTGCCGCCT	CCGTTTCCCG	GTGCCGGGAT	TCCGAGGAAG	AATGCACCGT	AGGAGGGGCA
17641	TGGCCGGCCA	CGGCCTGACG	GGCGGCATGC	GTCGTGCGCA	CCACCGGCGG	CGGCGCGCGT
17701	CGCACCGTCG	CATGCGCGGC	GGTATCCTGC	CCCTCCTTAT	TCCACTGATC	GCCGCGGCGA
17761	TTGGCGCCGT	GCCCCGAATT	GCATCCGTGG	CCTTGCAGGC	GCAGAGACAC	TGATTAAAAA
17821	CAAGTTGCAT	GTGGAAAAAT	CAAAATAAAA	AGTCTGGACT	CTCACGCTCG	CCTGGTCCCTG
17881	TAACTATTTT	GTAGAATGGA	AGACATCAAC	TTTGCGTCTC	TGGCCCCGCG	ACACGGCTCG
17941	CGCCCGTTCA	TGGGAAACTG	GCAAGATATC	GGCACCAGCA	ATATGAGCGG	TGGCGCCTTC
18001	AGCTGGGGCT	CGCTGTGGAG	CGGCATTAAA	AATTTTCGGT	CCACCGTTAA	GAACATATGGC
18061	AGCAAGGCCT	GGAACAGCAG	CACAGGCCAG	ATGCTGAGGG	ATAAGTTGAA	AGAGCAAAAT
18121	TTCCAACAAA	AGGTGGTAGA	TGGCCTGGCC	TCTGGCATT	GCGGGGTGGT	GGACCTGGCC
18181	AACCAGGCAG	TGCAAAATAA	GATTAACAGT	AAGCTTGATC	CCCGCCCTCC	CGTAGAGGAG
18241	CCTCCACCGG	CCGTGGAGAC	AGTGTCTCCA	GAGGGGCGTG	GCGAAAAGCG	TCCGCGCCCC
18301	GACAGGGAAG	AAACTCTGGT	GACGCAAATA	GACGAGCCTC	CCTCGTACGA	GGAGGCACTA
18361	AAGCAAGGCC	TGCCCACCAC	CCGTCCCATC	GCGCCCATGG	CTACCGGAGT	GCTGGGCCAG
18421	CACACACCCG	TAACGCTGGA	CCTGCCTCCC	CCCGCCGACA	CCCAGCAGAA	ACCTGTGCTG
18481	CCAGGCCCGA	CCGCCGTTGT	TGTAACCCGT	CCTAGCCGCG	CGTCCCTGCG	CCGCGCCGCC
18541	AGCGGTCCGC	GATCGTTGCG	GCCCCGTAGC	AGTGGCAACT	GGCAAAGCAC	ACTGAACAGC
18601	ATCGTGGGTC	TGGGGGTGCA	ATCCCTGAAG	CGCCGACGAT	GCTTCTGAAT	AGCTAACGTG
18661	TGCTATGTGT	GTCTATGTATG	CGTCCATGTC	GCCGCCAGAG	GAGCTGCTGA	GCCGCCGCGC
18721	GCCCGCTTTC	CAAGATGGCT	ACCCCTTCGA	TGATGCCGCA	GTGGTCTTAC	ATGCACATCT
18781	CGGGCCAGGA	CGCCTCGGAG	TACCTGAGCC	CCGGGCTGGT	GCAGTTTGCC	CGCGCCACCG
18841	AGACGTACTT	CAGCCTGAAT	AACAAGTTTA	GAAACCCAC	GGTGGCGCCT	ACGCACGACG
18901	TGACCACAGA	CCGGTCCCAG	CGTTTGACGC	TGCGGTTTAT	CCCTGTGGAC	CGTGAGGATA
18961	CTGCGTACTC	GTACAAGGCG	CGGTTTACCC	TAGCTGTGGG	TGATAACCGT	GTGCTGGACA
19021	TGGCTTCCAC	GTACTTTGAC	ATCCGCGGCG	TGCTGGACAG	GGGCCCTACT	TTTAAGCCCT
19081	ACTCTGGCAC	TGCCTACAAC	GCCCTGGCTC	CCAAGGGTGC	CCCAAATCCT	TGCGAATGGG
19141	ATGAAGCTGC	TACTGCTCTT	GAAATAAACC	TAGAAGAAGA	GGACGATGAC	AACGAAGACG
19201	AAGTAGACGA	GCAAGCTGAG	CAGCAAAAAA	CTCACGTATT	TGGGCAGGCG	CCTTATTCTG
19261	GTATAAATAT	TACAAAGGAG	GGTATTCAAA	TAGGTGTGCA	AGGTCAAACA	CCTAAATATG
19321	CCGATAAAAC	ATTTCAACCT	GAACCTCAAA	TAGGAGAATC	TCAGTGGTAC	GAAACTGAAA
19381	TTAATCATGC	AGCTGGGAGA	GTCTTAAAAA	AGACTACCCC	AATGAAACCA	TGTTACGGTT
19441	CATATGCAAA	ACCCACAAAT	GAAAATGGAG	GGCAAGGCAT	TCTTGTAAGG	CAACAAAATG
19501	GAAAGCTAGA	AAGTCAAGTG	GAAATGCAAT	TTTTCTCAAC	TACTGAGGCG	ACCGCAGGCA

19561	ATGGTGATAA	CTTGACTCCT	AAAGTGGTAT	TGTACAGTGA	AGATGTAGAT	ATAGAAACCC
19621	CAGACACTCA	TATTTCTTAC	ATGCCCACTA	TTAAGGAAGG	TAACCTCACGA	GAACCTAATGG
19681	GCCAACAATC	TATGCCCAAC	AGGCCTAATT	ACATTGCTTT	TAGGGACAAT	TTTATTGGTC
19741	TAATGTATTA	CAACAGCACG	GGTAATATGG	GTGTTCTGGC	GGGCCAAGCA	TCGCAGTTGA
19801	ATGCTGTTGT	AGATTTGCAA	GACAGAAACA	CAGAGCTTTC	ATACCAGCTT	TTGCTTGATT
19861	CCATTGGTGA	TAGAACCAGG	TACTTTTCTA	TGTGGAATCA	GGCTGTTGAC	AGCTATGATC
19921	CAGATGTTAG	AATTATTGAA	AATCATGGAA	CTGAAGATGA	ACTTCCAAAT	TACTGCTTTC
19981	CACTGGGAGG	TGTGATTAAT	ACAGAGACTC	TTACCAAGGT	AAAACCTAAA	ACAGGTCAGG
20041	AAAATGGATG	GGAAAAAGAT	GCTACAGAAT	TTTCAGATAA	AAATGAAATA	AGAGTTGGAA
20101	ATAATTTTGC	CATGGAAATC	AATCTAAATG	CCAACCTGTG	GAGAAATTTT	CTGTACTCCA
20161	ACATAGCGCT	GTATTTGCCC	GACAAGCTAA	AGTACAGTCC	TTCCAACGTA	AAAATTTCTG
20221	ATAACCCAAA	CACCTACGAC	TACATGAACA	AGCGAGTGGT	GGCTCCCGGG	TTAGTGGACT
20281	GCTACATTAA	CCTTGGAGCA	CGCTGGTCCC	TTGACTATAT	GGACAACGTC	AACCCATTTA
20341	ACCACCACCG	CAATGCTGGC	CTGCGCTACC	GCTCAATGTT	GCTGGGCAAT	GGTCGCTATG
20401	TGCCCTTCCA	CATCCAGGTG	CCTCAGAAGT	TCTTTGCCAT	TAAAAACCTC	CTTCTCCTGC
20461	CGGGCTCATA	CACCTACGAG	TGGAACCTTC	GGAAGGATGT	TAACATGGTT	CTGCAGAGCT
20521	CCCTAGGAAA	TGACCTAAGG	GTTGACGGAG	CCAGCATTAA	GTTTGATAGC	ATTTGCCTTT
20581	ACGCCACCTT	CTTCCCCATG	GCCCCAACA	CCGCCTCCAC	GCTTGAGGCC	ATGCTTAGAA
20641	ACGACACCAA	CGACCAGTCC	TTTAACGACT	ATCTCTCCGC	CGCCAACATG	CTCTACCCTA
20701	TACCCGCCAA	CGCTACCAAC	GTGCCATAT	CCATCCCCTC	CCGCAACTGG	CGCGCTTTCC
20761	GCGGCTGGGC	CTTCACGCGC	CTTAAGACTA	AGGAAACCCC	ATCACTGGGC	TCGGGCTACG
20821	ACCCTTATTA	CACCTACTCT	GGCTCTATAC	CCTACCTAGA	TGGAACCTTT	TACCTCAACC
20881	ACACCTTTAA	GAAGGTGGCC	ATTACCTTTG	ACTCTTCTGT	CAGCTGGCCT	GGCAATGACC
20941	GCCTGCTTAC	CCCCAACGAG	TTTGAAATTA	AGCGCTCAGT	TGACGGGGAG	GTTTACAACG
21001	TTGCCCAGTG	TAACATGACC	AAAGACTGGT	TCCTGGTACA	AATGCTAGCT	AACTACAACA
21061	TTGGCTACCA	GGGCTTCTAT	ATCCCAGAGA	GCTACAAGGA	CCGCATGTAC	TCCTTCTTTA
21121	GAAACTTCCA	GCCCATGAGC	CGTCAGGTGG	TGGATGATAC	TAAATACAAG	GACTACCAAC
21181	AGGTGGGCAT	CCTACACCAA	CACAACAAC	CTGGATTTGT	TGGCTACCTT	GCCCCACCA
21241	TGCGCGAAGG	ACAGGCCTAC	CCTGTAACT	TCCCCTATCC	GCTTATAGGC	AAGACCGCAG
21301	TTGACAGCAT	TACCCAGAAA	AAGTTTCTTT	GCGATCGCAC	CCTTTGGCGC	ATCCCATTCT
21361	CCAGTAACTT	TATGTCCATG	GGCGCACTCA	CAGACCTGGG	CCAAAACCTT	CTCTACGCCA
21421	ACTCCGCCCC	CGCGCTAGAC	ATGACTTTTG	AGGTGGATCC	CATGGACGAG	CCCACCCTTC
21481	TTTATGTTTT	GTTTGAAGTC	TTTGACTGGT	TCCGTGTGCA	CCGGCCGCAC	CGCGCGCTCA
21541	TCGAAACCGT	GTACCTGCGC	ACGCCCTTCT	CGGCCGGCAA	CGCCACAACA	TAAAGAAGCA
21601	AGCAACATCA	ACAACAGCTG	CCGCCATGGG	CTCCAGTGAG	CAGGAACCTGA	AAGCCATTGT
21661	CAAAGATCTT	GGTTGTGGGC	CATATTTTTT	GGGCACCTAT	GACAAGCGCT	TTCCAGGCTT
21721	TGTTTCTCCA	CACAAGCTCG	CCTGCGCCAT	AGTCAATACG	GCCGGTCGCG	AGACTGGGGG
21781	CGTACACTGG	ATGGCCTTTG	CCTGGAACCC	GCACTCAAAA	ACATGCTACC	TCTTTGAGCC
21841	CTTTGGCTTT	TCTGACCAGC	GACTCAAGCA	GGTTTACCAG	TTTGAGTACG	AGTCACTCCT
21901	GCGCCGTAGC	GCCATTGCTT	CTTCCCCCGA	CCGCTGTATA	ACGCTGGAAA	AGTCCACCCA
21961	AAGCGTACAG	GGGCCCAACT	CGGCCGCTTG	TGGACTATTG	TGCTGCATGT	TTCTCCACGC
22021	CTTTGCCAAC	TGGCCCCAAA	CTCCCATGGA	TCACAACCCC	ACCATGAACC	TTATTACCGG
22081	GGTACCCAAC	TCCATGCTCA	ACAGTCCCCA	GGTACAGCCC	ACCCTGCGTC	GCAACCAGGA
22141	ACAGCTCTAC	AGCTTCCTGG	AGCGCCACTC	GCCCTACTTC	CGCAGCCACA	GTGCGCAGAT
22201	TAGGAGCGCC	ACTTCTTTTT	GTCACCTTGA	AAACATGTAA	AAATAATGTA	CTAGAGACAC
22261	TTTCAATAAA	GGCAAATGCT	TTTATTTGTA	CACTCTCGGG	TGATTATTTA	CCCCCACCTT
22321	TGCCGTCTGC	GCCGTTTAAA	AATCAAAGGG	GTTCTGCCGC	GCATCGCTAT	GCGCCACTGG
22381	CAGGGACACG	TTGCGATACT	GGTGTTTAGT	GCTCCACTTA	AACTCAGGCA	CAACCATCCG
22441	CGGCAGCTCG	GTGAAGTTTT	CACCTCCACAG	GCTGCGCACC	ATCACCAACG	CGTTTAGCAG
22501	GTCGGGCGCC	GATATCTTGA	AGTCGCAGTT	GGGGCCTCCG	CCCTGCGCGC	GCGAGTTGCG
22561	ATACACAGGG	TTGCAGCACT	GGAACACTAT	CAGCGCCGGG	TGGTGCACGC	TGGCCAGCAC
22621	GCTCTTGTCG	GAGATCAGAT	CCGCGTCCAG	GTCCTCCGCG	TTGCTCAGGG	CGAACGGAGT
22681	CAACTTTGGT	AGCTGCCTTC	CCAAAAAGGG	CGCGTGCCCA	GGCTTTGAGT	TGCACTCGCA
22741	CCGTAGTGGC	ATCAAAAGGT	GACCGTGCCC	GGTCTGGGCG	TTAGGATACA	GCGCCTGCAT
22801	AAAAGCCTTG	ATCTGCTTAA	AAGCCACCTG	AGCCTTTGCG	CCTTCAGAGA	AGAACATGCC
22861	GCAAGACTTG	CCGGAAAACT	GATTGGCCGG	ACAGGCCGCG	TCGTGCACGC	AGCACCTTGC
22921	GTCGGTGTTG	GAGATCTGCA	CCACATTTCG	GCCCCACCGG	TTCTTCACGA	TCTTGGCCTT

22981	GCTAGACTGC	TCCTTCACGC	CGCGCTGCC	GTTTTGCTC	GTCACATCCA	TTTCAATCAC
23041	GTGCTCCTTA	TTTATCATAA	TGCTTCCGTG	TAGACACTTA	AGCTCGCCTT	CGATCTCAGC
23101	GCAGCGGTGC	AGCCACAACG	CGCAGCCCGT	GGGCTCGTGA	TGCTTGTAGG	TCACCTCTGC
23161	AAACGACTGC	AGGTACGCCT	GCAGGAATCG	CCCCATCATC	GTCACAAAGG	TCCTTGTTGCT
23221	GGTGAAGGTC	AGCTGCAACC	CGCGGTGCTC	CTCGTTTCAGC	CAGGTCTTGC	ATACGGCCGC
23281	CAGAGCTTCC	ACTTGGTFCAG	GCAGTAGTTT	GAAGTTTCGCC	TTTAGATCGT	TATCCACGTG
23341	GTACTTGTCC	ATCAGCGCGC	GCGCAGCCTC	CATGCCCTTC	TCCCACGCGC	ACACGATCGG
23401	CACACTCAGC	GGGTTCATCA	CCGTAATTTT	ACTTTCCGCT	TCGCTGGGCT	CTTCTCTTTC
23461	CTCTTGCGTC	CGCATACCAC	GCGCCACTGG	GTCGTCTTCA	TTCAGCCGCC	GCACCTGTGCG
23521	CTTACCTCCT	TTGCCATGCT	TGATTAGCAC	CGGTGGGTTG	CTGAAACCCA	CCATTTGTAG
23581	CGCCACATCT	TCTCTTTCTT	CCTCGCTGTC	CACGATTACC	TCTGGTGTATG	GCGGGCGCTC
23641	GGGCTTGGGA	GAAGGGCGCT	TCTTTTTCTT	CTTGGGCGCA	ATGGCCAAAT	CCGCCGCCGA
23701	GGTCGATGGC	CGCGGGCTGG	GTGTGCGCGG	CACGAGCGCG	TCTTGTGTATG	AGTCTTCTTC
23761	GTCTCTCGAC	TCGATACGCC	GCCTCATCCG	CTTTTTTGGG	GGCGCCCGGG	GAGGCGGCGG
23821	CGACGGGGAC	GGGGACGACA	CGTCCTCCAT	GGTTGGGGGA	CGTCGCGCCG	CACCGCGTCC
23881	GCGCTCGGGG	GTGGTTTCGC	GCTGCTCCTC	TTCCCGACTG	GCCATTTCTT	TCTCCTATAG
23941	GCAGAAAAAG	ATCATGGAGT	CAGTCGAGAA	GAAGGACAGC	CTAACCGCCC	CCTCTGAGTT
24001	CGCCACCACC	GCCTCCACCG	ATGCCGCCAA	CGCGCCTACC	ACCTTCCCCG	TCGAGGCACC
24061	CCCGCTTGAG	GAGGAGGAAG	TGATTATCGA	CGAGGACCCA	GGTTTTGTAA	CGGAAGACGA
24121	CGAGGACCGC	TCAGTACCAA	CAGAGGATAA	AAAGCAAGAC	CAGGACAACG	CAGAGGCAAA
24181	CGAGGAACAA	GTCGGGCGGG	GGGACGAAAG	GCATGGCGAC	TACCTAGATG	TGGGAGACGA
24241	CGTGCTGTTG	AAGCATCTGC	AGCGCCAGTG	CGCCATTATC	TGCGACGCGT	TGCAAGAGCG
24301	CAGCGATGTG	CCCCTCGCCA	TAGCGGATGT	CAGCCTTGCC	TACGAACGCC	ACCTATTCTC
24361	ACCGCGCGTA	CCCCCAAAC	GCCAAGAAAA	CGGCACATGC	GAGCCCAACC	CGCGCCTCAA
24421	CTTCTACCCC	GTATTTGCGG	TGCCAGAGGT	GCTTGCCACC	TATCACATCT	TTTTCCAAAA
24481	CTGCAAGATA	CCCCTATCCT	GCCGTGCCAA	CCGCAGCCGA	GCGGACAAGC	AGCTGGCCTT
24541	GCGGCAGGGC	GCTGTCATAC	CTGATATCGC	CTCGCTCAAC	GAAGTGCCAA	AAATCTTTGA
24601	GGTCTTTGGA	CGCGACGAGA	AGCGCGCGG	AAACGCTCTG	CAACAGGAAA	ACAGCGAAAA
24661	TGAAGTTCAC	TCTGGAGTGT	TGGTGGAAGT	CGAGGCTGAC	AACCGCGGCC	TAGCCGTACT
24721	AAAACGCAGC	ATCGAGGTCA	CCCCTTTGCT	CTACCCGGCA	CTTAACCTAC	CCCCCAAGGT
24781	CATGAGCACA	GTCATGAGTG	AGCTGATCGT	GCGCCGTGCG	CAGCCCCTGG	AGAGGGATGC
24841	AAATTTGCAA	GAACAAACAG	AGGAGGGCCT	ACCCGAGTTT	GGCGACGAGC	AGCTAGCGCG
24901	CTGGCTTCAA	ACGCGCGAGC	CTGCCGACTT	GGAGGAGCGA	CGCAAACATA	TGATGGCCGC
24961	AGTGCTCGTT	ACCGTGAGAG	TTGAGTGCAT	GCAGCGGTTT	TTTGCTGACC	CGGAGATGCA
25021	GCGCAAGCTA	GAGGAAACAT	TGCACTACAC	CTTTGACACG	GGCTACGTAC	GCCAGGCCTG
25081	CAAGATCTCC	AACGTGGAGC	TCTGCAACCT	GGTCTCCTAC	CTTGGAATTT	TGCACGAAAA
25141	CCGCCTTGGG	CAAAACGTGC	TTCAATCCAC	GCTCAAGGGC	GAGGCGCGCC	GCGCATCGT
25201	CCGCGACTGC	GTTTACTTAT	TTCTATGCTA	CACCTGGCAG	ACGGCCATGG	GCGTTTGCGA
25261	GCAGTGCTTG	GAGGAGTGCA	ACCTCAAGGA	GCTGCAGAAA	CTGCTAAAGC	AAAACCTGAA
25321	GGACCTATGG	ACGGCCTTCA	ACGAGCGCTC	CGTGGCCGCG	CACCTGGCGG	ACATCATTTT
25381	CCCCGAACGC	CTGCTTAAAA	CCCTGCAACA	GGGTCTGCCA	GACTTCACCA	GTCAAAGCAT
25441	GTTGCAGAAC	TTTAGGAACT	TTATCCTAGA	GCGCTCAGGA	ATCTTGCCCG	CCACCTGCTG
25501	TGCACTTCCT	AGCGACTTTG	TGCCCATTA	GTACCGCGAA	TGCCCTCCGC	CGCTTTGGGG
25561	CCACTGCTAC	CTTCTGCAGC	TAGCCAACTA	CCTTGCCCTAC	CACTCTGACA	TAATGGAAGA
25621	CGTGAGCGGT	GACGGTCTAC	TGGAGTGTC	CTGTGCTGTC	AACCTATGCA	CCCCGCACCG
25681	CTCCCTGGTT	TGCAATTTCG	AGCTGCTTAA	CGAAAGTCAA	ATTATCGGTA	CCTTTGAGCT
25741	GCAGGGTCCC	TCGCCCTGAG	AAAAGTCCGC	GGCTCCGGGG	TTGAAACTCA	CTCCGGGGCT
25801	GTGGACGTCG	GCTTACCTTC	GCAAATTTGT	ACCTGAGGAC	TACCACGCCC	ACGAGATTAG
25861	GTTCTACGAA	GACCAATCCC	GCCCGCCAAA	TGCGGAGCTT	ACCGCCTGCG	TCATTACCCA
25921	GGGCCACATT	CTTGGCCAAT	TGCAAGCCAT	CAACAAAGCC	CGCCAAGAGT	TTCTGCTACG
25981	AAAGGGACGG	GGGGTTTACT	TGGACCCCCA	GTCCGGCGAG	GAGCTCAACC	CAATCCCCCC
26041	GCCGCCGCAG	CCCTATCAGC	AGCAGCCGCG	GGCCTTGCT	TCCCAGGATG	GCACCCAAAA
26101	AGAAGCTGCA	GCTGCCGCCG	CCACCCACGG	ACGAGGAGGA	ATACTGGGAC	AGTCAGGCAG
26161	AGGAGGTTTT	GGACGAGGAG	GAGGAGGACA	TGATGGAAGA	CTGGGAGAGC	CTAGACGAGG
26221	AAGCTTCCGA					

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26401	CCGGTAAGTC	CAAGCAGCCG	CCGCCGTTAG	CCCAAGAGCA	ACAACAGCGC	CAAGGCTACC
26461	GCTCATGGCG	CGGGCACAAG	AACGCCATAG	TTGCTTGCTT	GCAAGACTGT	GGGGGCAACA
26521	TCTCCTTCGC	CCGCCGCTTT	CTTCTCTACC	ATCACGGCGT	GGCCTTCCCC	CGTAACATCC
26581	TGCATTACTA	CCGTCATCTC	TACAGCCCAT	ACTGCACCGG	CGGCAGCGGC	AGCGGCAGCA
26641	ACAGCAGCGG	CCACACAGAA	GCAAAGGCGA	CCGGATAGCA	AGACTCTGAC	AAAGCCCAAG
26701	AAATCCACAG	CGGCGGCAGC	AGCAGGAGGA	GGAGCGCTGC	GTCTGGCGCC	CAACGAACCC
26761	GTATCGACCC	GCGAGCTTAG	AAACAGGATT	TTTCCCCTCT	TGTATGCTAT	ATTTCACAG
26821	AGCAGGGGCC	AAGAACAAGA	GCTGAAAATA	AAAAACAGGT	CTCTGCGATC	CCTCACCCGC
26881	AGCTGCCTGT	ATCACAAAAG	CGAAGATCAG	CTTCGGCGCA	CGCTGGAAGA	CGCGGAGGCT
26941	CTCTTCAGTA	AATACTGCGC	GCTGACTCTT	AAGGACTAGT	TTTCGCGCCT	TTCTCAAATT
27001	TAAGCGCGAA	AACACTCGTC	TCTCCAGCGG	CCACACCCGG	CGCCAGCACC	TGTCGTACAG
27061	GCCATTATGA	GCAAGGAAAT	TCCCACGCC	TACATGTGGA	GTTACCAGCC	ACAAATGGGA
27121	CTTGCGGCTG	GAGCTGCCCA	AGACTACTCA	ACCCGAATAA	ACTACATGAG	CGCGGGACCC
27181	CACATGATAT	CCCGGGTCAA	CGGAATCCGC	GCCCACCGAA	ACCGAATTCT	CTTGGAACAG
27241	GCGGCTATTA	CCACCACACC	TCGTAATAAC	CTTAATCCCC	GTAGTTGGCC	CGCTGCCCTG
27301	GTGTACCAGG	AAAGTCCCGC	TCCCACCACT	GTGGTACTTC	CCAGAGACGC	CCAGGCCGAA
27361	GTTTCAGATG	CTAACTCAGG	GGCGCAGCTT	GCGGGCGGCT	TTTCGTACAG	GGTGCGGTCT
27421	CCCGGGCAGG	GTATAACTCA	CCTGACAATC	AGAGGGCGAG	GTATTCAGCT	CAACGACGAG
27481	TCGGTGAGCT	CCTCGCTTGG	TCTCCGTCCG	GACGGGACAT	TTTCAGATCG	CGGCGCCGGC
27541	CGTCCCTTCAT	TCACGCCTCG	TCAGGCAATC	CTAACTCTGC	AGACCTCGTC	CTCTGAGCCG
27601	CGCTCTGGAG	GCATTGGAAC	TCTGCAATTT	ATTGAGGAGT	TTGTGCCATC	GTCTCACTTT
27661	AACCCCTTCT	CGGGACCTCC	CGGCCACTAT	CCGGATCAAT	TTATTCTTAA	CTTTGACGCG
27721	GTAAAGGACT	CGGCGGACGG	CTACGACTGA	ATGTTAAGTG	GAGAGGCAGA	GCAACTGCGC
27781	CTGAAACACC	TGGTCCACTG	TCGCCGCCAC	AAGTGCTTTG	CCCGCGACTC	CGGTGAGTTT
27841	TGCTACTTTG	AATTGCCCCG	GGATCATATC	GAGGGCCCGG	CGCACGGCGT	CCGGCTTACC
27901	GCCCAGGGAG	AGCTTGCCCG	TAGCCTGATT	CGGGAGTTTA	CCCAGCGCCC	CCTGCTAGTT
27961	GAGCGGGACA	GGGGACCTTG	TGTTCTCACT	GTGATTTGCA	ACTGTCCTAA	CCTTGATTAA
28021	CATCAAGATC	TTTGTTGCCA	TCTCTGTGCT	GAGTATAATA	AATACAGAAA	TTAAATATA
28081	CTGGGGCTCC	TATCGCCATC	CTGTAAACGC	CACCGTCTTC	ACCCGCCCAA	GCAAACCAAG
28141	GCGAACCTTA	CCTGGTACTT	TTAACATCTC	TCCCTCTGTG	ATTTACAACA	GTTTCAACCC
28201	AGACGGAGTG	AGTCTACGAG	AGAACCTCTC	CGAGCTCAGC	TACTCCATCA	GAAAAAACAC
28261	CACCTCCTT	ACCTGCCGGG	AACGTACGAG	TGCGTCACCG	GCCGCTGCAC	CACACCTACC
28321	GCCTGACCGT	AAACCAGACT	TTTTCCGGAC	AGACCTCAAT	AACTCTGTTT	ACCAGAACAG
28381	GAGGTGAGCT	TAGAAAACCC	TTAGGGTATT	AGGCCAAAGG	CGCAGCTACT	GTGGGGTTTA
28441	TGAACAATTC	AAGCAACTCT	ACGGGCTATT	CTAATTCAGG	TTTCTCTAGA	AGTCAGGCTT
28501	CCTGGATGTC	AGCATCTGAC	TTTGCCAGC	ACCTGTCCCG	CGGATTTGTT	CCAGTCCAAC
28561	TACAGCGACC	CACCCTAACA	GAGATGACCA	ACACAACCAA	CGCGGCCGCC	GCTACCGGAC
28621	TTACATCTAC	CACAAATACA	CCCCAAGTTT	CTGCCTTTGT	CAATAACTGG	GATAACTTGG
28681	GCATGTGGTG	GTTCTCCATA	GCGCTTATGT	TTGTATGCCT	TATTATTATG	TGGCTCATCT
28741	GCTGCCTAAA	GCGCAAACGC	GCCCGACCAC	CCATCTATAG	TCCCATCATT	GTGCTACACC
28801	CAAACAATGA	TGGAATCCAT	AGATTGGACG	GACTGAAACA	CATGTTCTTT	TCTCTTACAG
28861	TATGATTAAA	TGAGATCTAG	AAATGGACGG	AATTATTACA	GAGCAGCGCC	TGCTAGAAAG
28921	ACGCAGGGCA	GCGGCCGAGC	AACAGCGCAT	GAATCAAGAG	CTCCAAGACA	TGGTTAACTT
28981	GCACCAAGTG	AAAAGGGGTA	TCTTTTGTCT	GGTAAAGCAG	GCCAAAGTCA	CCTACGACAG
29041	TAATACCACC	GGACACCGCC	TTAGCTACAA	GTGCCAACC	AAGCGTCAGA	AATTGGTGGT
29101	CATGGTGGGA	GAAAAGCCCA	TTACCATAAC	TCAGCACTCG	GTAAGAAACCG	AAGGCTGCAT
29161	TCACTCACCT	TGTCAAGGAC	CTGAGGATCT	CTGCACCTT	ATTAAGACCC	TGTGCGGTCT
29221	CAAAGATCTT	ATTCCCTTTA	ACTAATAAAA	AAAAATAATA	AAGCATCACT	TACTTAAAT
29281	GAGTTAGCAA	ATTTCTGTCC	AGTTTATTCA	GCAGCACCTC	CTTGCCCTCC	TCCCAGCTCT
29341	CGTATTGCAG	CTTCTCTCTG	GCTGCAAACT	TTCTCCACAA	TCTAAATGGA	ATGTCAGTTT
29401	CCTCCTGTTT	CTGTCCATCC	GCACCCACTA	TCTTCATGTT	GTTGCAGATG	AAGCGCGCAA
29461	GACCGTCTGA	AGATACCTTC	AACCCCGTGT	ATCCATATGA	CACGGAAACC	GGTCTCCAA
29521	CTGTGCCTTT	TCTTACTCCT	CCCTTTGTAT	CCCCAATGG	GTTTCAAGAG	AGTCCCCCTG
29581	GGGTACTCTC	TTTGCGCCTA	TCCGAACCTC	TAGTTACCTC	CAATGGCATG	CTTGCGCTCA
29641	AAATGGGCAA	CGGCCTCTCT	CTGGACGAGG	CCGGCAACCT	TACCTCCCAA	AATGTAACCA
29701	CTGTGAGCCC	ACCTCTCAAA	AAAACCAAGT	CAAACATAAA	CCTGGAAATA	TCTGCACCCC
29761	TCACAGTTAC	CTCAGAAGCC	CTAACTGTGG	CTGCCGCCGC	ACCTCTAATG	GTGCGGGGCA

FIGURE 23
(SHEET 9)

29821	ACACACTCAC	CATGCAATCA	CAGGCCCCGC	TAACCGTGCA	CGACTCCAAA	CTTAGCATTG
29881	CCACCCAAGG	ACCCCTCACA	GTGTCAGAAG	GAAAGCTAGC	CCTGCAAACA	TCAGGCCCCC
29941	TCACCACCAC	CGATAGCAGT	ACCCTTACTA	TCAGTGCCTC	ACCCCTCTA	ACTACTGCCA
30001	CTGGTAGCTT	GGGCATTGAC	TTGAAAGAGC	CCATTTTATAC	ACAAAATGGA	AAACTAGGAC
30061	TAAAGTACGG	GGCTCCTTTG	CATGTAACAG	ACGACCTAAA	CACTTTTGACC	GTAGCAACTG
30121	GTCCAGGTGT	GACTATTAAT	AATAC'TTCCT	TGCAAACTAA	AGTTACTGGA	GCCTTGGGTT
30181	TTGATTACAA	AGGCAATATG	CAACTTAATG	TAGCAGGAGG	ACTAAGGATT	GATTCTCAAA
30241	ACAGACGCCT	TATACTTGAT	GTTAGTTATC	CGTTTGATGC	TCAAAACCAA	CTAAATCTAA
30301	GACTAGGACA	GGGCCCTCTT	TTTATAAACT	CAGCCCACAA	CTTGGATATT	AACTACAACA
30361	AAGGCCTTTA	CTTGTTTACA	GCTTCAAACA	ATTCCAAAAA	GCTTGAGGTT	AACCTAAGCA
30421	CTGCCAAGGG	GTTGATGTTT	GACGCTACAG	CCATAGCCAT	TAATGCAGGA	GATGGGCTTG
30481	AATTTGGTTC	ACCTAATGCA	CCAAACACAA	ATCCCCTCAA	AACAAAAATT	GGCCATGGCC
30541	TAGAATTTGA	TTCAAACAAG	GCTATGGTTC	CTAAACTAGG	AACTGGCCTT	AGTTTGTACA
30601	GCACAGGTGC	CATTACAGTA	GGAAACAAAA	ATAATGATAA	GCTAACTTTG	TGGACCACAC
30661	CAGCTCCATC	TCCTAACTGT	AGACTAAATG	CAGAGAAAGA	TGCTAAACTC	ACTTTGGTCT
30721	TAACAAAATG	TGGCAGTCAA	ATACTTGCTA	CAGTTTCAGT	TTTGGCTGTT	AAAGGCAGTT
30781	TGGCTCCAAT	ATCTGGAACA	GTTCAAAGTG	CTCATCTTAT	TATAAGATTT	GACGAAAATG
30841	GAGTGTACT	AAACAATTCC	TTCCTGGACC	CAGAATATTG	GAAC'TTTAGA	AATGGAGATC
30901	TTACTGAAGG	CACAGCCTAT	ACAAACGCTG	TTGGATTTAT	GCCTAACCTA	TCAGCTTATC
30961	CAAAATCTCA	CGGTAAAACT	GCCAAAAGTA	ACATTGTGAG	TCAAGTTTAC	TTAAACGGAG
31021	ACAAAAC'TAA	ACCTGTAACA	CTAACCATTA	CACTAAACGG	TACACAGGAA	ACAGGAGACA
31081	CAACTCCAAG	TGCATACTCT	ATGTCA'TTTT	CATGGGACTG	GTCTGGCCAC	AACTACATTA
31141	ATGAAATATT	TGCCACATCC	TCTTACACTT	TTTCATACAT	TGCCCAAGAA	TAAAGAATCG
31201	TTTGTGTTAT	GTTTCAACGT	GTTTATTTT	CAATTGCAGA	AAATTTCAAG	TCATTTTTC
31261	TTCAGTAGTA	TAGCCCCACC	ACCACATAGC	TTATACAGAT	CACCGTACCT	TAATCAAACCT
31321	CACAGAACCC	TAGTATTCAA	CCTGCCACCT	CCCTCCCAAC	ACACAGAGTA	CACAGTCCTT
31381	TCTCCCCGGC	TGGCCTTAAA	AAGCATCATA	TCATGGGTAA	CAGACATATT	CTTAGGTGTT
31441	ATATTCCACA	CGGTTTCTTG	TCGAGCCAAA	CGCTCATCAG	TGATATTAAT	AAACTCCCCG
31501	GGCAGCTCAC	TTAAGTTCAT	GTCGCTGTCC	AGCTGCTGAG	CCACAGGCTG	CTGTCCAACCT
31561	TGCGGTTGCT	TAACGGGCGG	CGAAGGAGAA	GTCCACGCCT	ACATGGGGGT	AGAGTCATAA
31621	TCGTGCATCA	GGATAGGGCG	GTGGTGTGTC	AGCAGCGCGC	GAATAAACTG	CTGCCGCCCG
31681	CGCTCCGTCC	TGCAGGAATA	CAACATGGCA	GTGGTCTCCT	CAGCGATGAT	TCGCACCGCC
31741	CGCAGCATAA	GGCGCCTTGT	CCTCCGGGCA	CAGCAGCGCA	CCCTGATCTC	ACTTAAATCA
31801	GCACAGTAAC	TGCAGCACAG	CACCACAATA	TTGTTCAAAA	TCCCACAGTG	CAAGGCGCTG
31861	TATCCAAAGC	TCATGGCGGG	GACCACAGAA	CCCACGTGGC	CATCATACCA	CAAGCGCAGG
31921	TAGATTAAGT	GGCGACCCCT	CATAAACACG	CTGGACATAA	ACATTACCTC	TTTTTGGCATG
31981	TTGTAAATCA	CCACCTCCCG	GTACCATATA	AACCTCTGAT	TAAACATGGC	GCCATCCACC
32041	ACCATCCTAA	ACCAGCTGGC	CAAAACCTGC	CCGCCGGCTA	TACACTGCAG	GGAACCGGGA
32101	CTGGAACAAT	GACAGTGGAG	AGCCCAGGAC	TCGTAACCAT	GGATCATCAT	GCTCGTCATG
32161	ATATCAATGT	TGGCACAACA	CAGGCACACG	TGCATACACT	TCCTCAGGAT	TACAAGCTCC
32221	TCCCGCCTTA	GAACCATATC	CCAGGGAACA	ACCCATTCCCT	GAATCAGCGT	AAATCCACAA
32281	CTGCAGGGAA	GACCTCGCAC	GTAAC'TCACG	TTGTGCATTG	TCAAAGTGT	ACATTCCGGC
32341	AGCAGCGGAT	GATCCTCCAG	TATGGTAGCG	CGGGTTTCTG	TCTCAAAAGG	AGGTAGACGA
32401	TCCCTACTGT	ACGGAGTGCG	CCGAGACAAC	CGAGATCGTG	TTGGTTCGTAG	TGTCATGCCA
32461	AATGGAACGC	CGGACGTAGT	CATATTTCTT	GAAGCAAAAC	CAGGTGCGGG	CGTGACAAAC
32521	AGATCTGCGT	CTCCGGTCTC	GCCGCTTAGA	TCGCTCTGTG	TAGTAGTTGT	AGTATATCCA
32581	CTCTCTCAAA	GCATCCAGGC	GCCCCCTGGC	TTCGGGTTCT	ATGTAAACTC	CTTCATGCGC
32641	CGCTGCCCTG	ATAACATCCA	CCACCGCAGA	ATAAGCCACA	CCCAGCCAAC	CTACACATTTC
32701	GTTCTGCGAG	TCACACACGG	GAGGAGCGGG	AAGAGCTGGA	AGAACCATGT	TTTTTTTTTTT
32761	ATTCCAAAAG	ATTATCCAAA	ACCTCAAAAT	GAAGATCTAT	TAAGTGAACG	CGCTCCCCCTC
32821	CGGTGGCGTG	GTCAAAC'TCT	ACAGCCAAAG	AACAGATAAT	GGCATT'TGTA	AGATGTTGCA
32881	CAATGGCTTC	CAAAAGGCAA	ACGGCCCTCA	CGTCCAAGTG	GACGTAAAGG	CTAAACCCCTT
32941	CAGGGTGAAT	CTCCTCTATA	AACATTCCAG	CACCTTCAAC	CATGCCCAAA	TAATTCTCAT
33001	CTCGCCACCT	TCTCAATATA	TCTCTAAGCA	AATCCCGAAT	ATTAAGTCCG	GCCATTGTAA
33061	AAATCTGTCT	CAGAGCGCCC	TCCACCTTCA	GCCTCAAGCA	GCGAATCATG	ATTGCAAAAA
33121	TTCAGGTTCC	TCACAGACCT	GTATAAGATT	CAAAAGCGGA	ACATTAACAA	AAATACCGCG
33181	ATCCCGTAGG	TCCCTTCGCA	GGGCCAGCTG	AACATAATCG	TGCAGGTCTG	CACGGACCAG

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